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CONTROLLED FLUID THERAPY

With Hematocrit, Specific Gravity, and Plasma Protein Determinations

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WITH the increasing consciousness of the importance of fluids has come an increasing demand for more rapid, accurate, objective methods of determining the fluid requirements of acutely ill patients and for maintaining water balance when once established.

We have found that 4 relatively simple tests give much useful information concerning the state of hydration in seriously ill surgical patients (26). These are: (1) Determination of the percentage of cells in venous blood by means of a hematocrit; (2) determination of the specific gravity of the whole blood; (3) determination of the specific gravity of the plasma, and (4) calculation of the plasma protein content by means of a simple formula. The merit of these combined tests lies in the speed with which they can be done, the accuracy with which results can be reproduced, the small amount of equipment necessary, and the ease with which the technique may be mastered.

The danger lies in attempting to interpret these findings without a clear clinical picture of the patient. This approach to a difficult problem is presented as an aid to, not as a substitute for, already well established, diagnostic procedures.

The opinions expressed here have grown out of 3 years' experience. Because of the numerous requests as to both methods and interpretation (27, 28), each of the tests will be considered separately in some detail.

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THE HEMATOCRIT

In 1885, Professor Blix presented at Upsala the first "haematokrit." It was modeled after the "laktokrit" used in the dairy industry. Employing this method, Hedin, in 1891, reported an average cell volume for adult males to be 48.0 per cent, and for adult females, 43.3 per cent. In the next 10 years there were many modifications. Capps, in 1903, introduced this work to America.

Haden (11), in 1923, popularized the large hematocrit tube in contradistinction to the capillary type and stressed the importance of using isotonic solutions of the various anticoagulants. Van Allen, in 1925, published a comprehensive résumé on the hematocrit method in experimental work. Wintrobe and Miller then introduced a 4 cubic centimeter graduate tube, made from a Mohr pipette; they employed potassium oxalate as the anticoagulant and allowed 6.7 per cent for cell

shrinkage. The publication of Haden (12), in 1930, is very complete and for further details this article is recommended.

In 1929, Sanford and Magath modified the Haden hematocrit. It is this tube which we prefer because it can be spun in any routine laboratory centrifuge, can be cleaned easily, and, being made of heavy glass, its durability is enhanced.

Anticoagulant. Heparin is the anticoagulant recommended for hematocrit determinations (8). It is an active fraction of the naturally occurring anticoagulant which was first isolated in Howell's laboratory by McLean in 1916. The heparin now employed is the sodium salt as prepared in the Connaught Laboratories of Toronto University, Canada. One milligram of the powder is sufficient anticoagulant for the blood in a Sanford-Magath hematocrit tube.

Directions for taking blood and reading the hematocrit. 1. Draw blood from vein with a sterile dry syringe without the use of a tourniquet if possible; or, if a tourniquet is used, release it and wait a minute for blood to recirculate before drawing the sample.

2. Gently introduce from 5 to 6 cubic centimeters into a Sanford-Magath hematocrit tube containing the proper amount of heparin. Avoid air bubbles.

3. Gently invert the tube two or three times to mix the blood with the anticoagulant.

4. Cork with rubber stopper (no-air No. 5), counter balance, and spin in centrifuge for 1 hour at 2,500 revolutions a minute (in emergency cases 15 minutes is sufficient).

5. Record the level of cells and the level of total blood sample.

6. To determine cell volume, divide the cells by the total volume. In normal blood the white cells form just a thin layer on top of the red cells, and it is not worth-while to attempt differentiation. In severe infections, however, usually associated with some degree of anemia, it is worth-while to measure the cell volume of the erythrocytes and leucocytes separately by dividing the volumes of each by the total blood volume in the sample.

7. Hemolysis in the plasma is most often caused by the use of a *wet syringe*, and less often by too vigorous shaking. The presence of a thin filament extending from bottom of the meniscus to top of the cells indicates a fibrinogen-fibrin mixture due either to insufficient anticoagulant or incomplete mixing. It decreases the cell volume somewhat and the plasma specific gravity determinations, but neither is significant for clinical purposes.

Normal values (Fig. 1). The normal cell volume values for the male range between 42 and 50 per cent, the average being approximately 46; while those for a female have a range of 39 to 43 per cent, with an average of 41.

SPECIFIC GRAVITY

The earliest investigator of blood specific gravity was Robert Boyle who, in 1684, showed that both serum and whole blood were heavier than water. Jurin, in 1719, measured their weights more accurately and reported the specific gravity of the blood as 1.053 and that of the serum as 1.030. Sir John Davy, in 1839, determined by pycnometry the specific gravity of the whole blood and quoted freely from the earlier work of John Hunter who showed that specific gravity was high in the morning, high in inflammation, and high in dehydration.

Roy, in 1884, reported a simplified method of weighing blood. E. Lloyd Jones (16, 17), in 1887, and later in 1891, used this method and published observations which are still of outstanding value for specific gravity determinations in both health and disease.

Hammerschlag compiled a very complete summary and introduced the use of benzol and chloroform as a modification of the Roy method. Sherrington and Copeman observed that a fall in blood pressure during a long experiment or operation was accompanied by a fall in specific gravity of venous blood; hemorrhage was followed by a rapid fall; while vasoconstriction as seen in shock caused an early rise in the specific gravity of peripheral blood. Rogers showed the value of these tests in treating the severe dehydration of cholera.

In 1924, Barbour and Hamilton (2, 3, 4) presented a means for determining the specific gravity of body fluids which eliminated many disadvantages of the older methods (Fig. 2). The principle based on Stokes' law takes advantage of the fact that the time required for a drop of known volume to fall a fixed distance through an immiscible fluid is governed by the density of the drop and other factors, such as temperature, which can be controlled easily. It has been shown that differences of 0.2 of 1 per cent in weight are demonstrable, and that specific gravities may be reproduced with an accuracy of 0.0001.

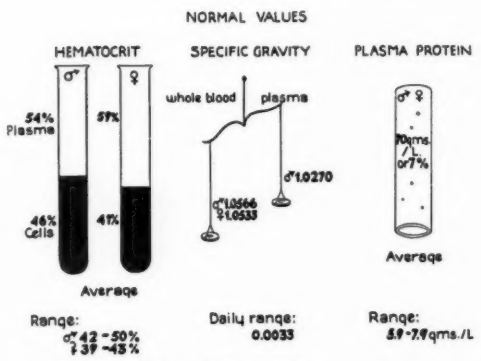

Fig. 1. Normal values.

Stebbins and Leake, in 1927, and later Polowe, and Bender and Polowe employed this method for clinical purposes. Guthrie, in 1932, reported that when compared with other methods for evaluation of blood conditions it stood first from every standpoint. For those unacquainted with the values of whole blood specific gravity, he offered the following simplified comparative table of approximate values:

Specific gravity	Hemoglobin	Hematocrit per cent cells	Red blood count
1.030	0	0	0
1.035	20	10	1,000,000
1.040	40	20	2,000,000
1.045	60	30	3,000,000
1.050	80	40	4,000,000
1.055	100	50	5,000,000
1.060	120	60	6,000,000

It is a modification of the Barbour and Hamilton apparatus[1] which we use routinely in the determination of whole blood, serum, and plasma specific gravity.

Normal values. In the male the average value of peripheral blood is 1.0566 and in the female 1.0533. A swing of 0.0033 occurs daily; the blood is more concentrated in the morning.

Practical directions for determination of the specific gravity of whole blood (Fig. 3). 1. Observe and record temperature of water bath in which the falling drop tubes are immersed.

[1]Complete sets may be obtained from Eimer and Amend, New York City, or LaMotte Chemical Company, Baltimore, Maryland.

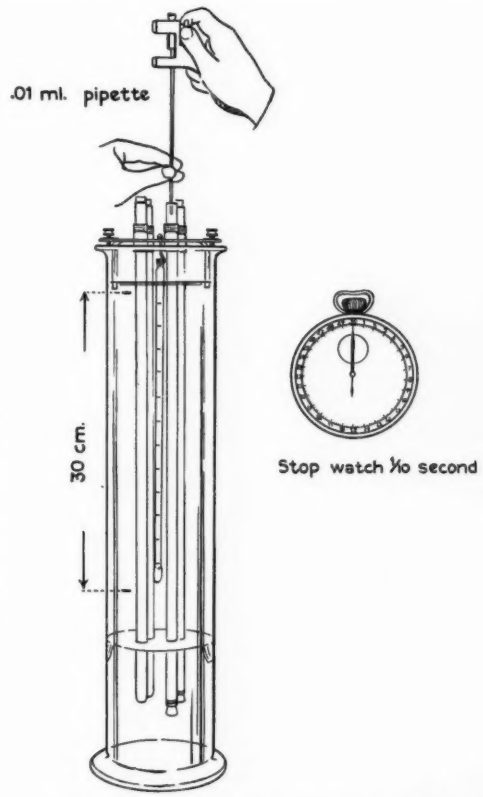

Fig. 2. Modified Barbour and Hamilton falling drop apparatus with Guthrie pipette holder. From the graduated pipette 0.01 ml. of plasma is released into a tube containing a mixture of bromobenzene and xylene and timed with a stop watch in its course between the two marks on the tube. The same process is carried out with a solution of known specific gravity and the weight of the unknown calculated (4).

2. Set stop watch at zero time.
3. Remove cork from the bromobenzene tube to be used.
   B_1 for normal and concentrated bloods (6).
   Specific gravity 1.0530.
   B_2 for anemic bloods.
   Specific gravity 1.0430.
4. Load pipette into holder.
5. Puncture finger; wipe away first drop; draw up blood into the calibrated pipette from a free bleeding, puncture wound to the second mark. (The pipette is made to deliver 2 drops of .01 milliliter each.)
6. Quickly (for no anticoagulant is used with whole blood) wipe the outside of the pipette with gauze, bring the drop of blood even with the middle

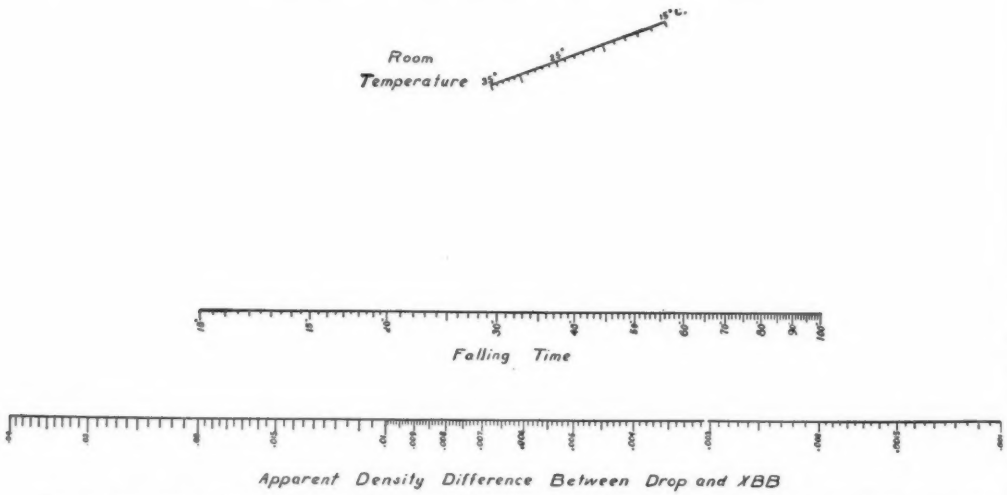

Fig. 3. Nomogram. Directions: (1) Insert pin with thread attached on the "temperature scale" at the recorded temperature of the water bath; (2) draw thread across the "falling time" scale at the correct time in seconds that it took the plasma to fall 30 centimeters; (3) read off the figure which the thread bisects on the *apparent density difference* scale. (This gives the difference in density between the xylene bromobenzene mixture and the plasma); (4) repeat same steps for the standard; (5) calculate specific gravity as directed. (Courtesy of H. G. Barbour, J. Am. M. Ass., 1927, 88: 91–94.)

mark on the pipette, insert the tip of the pipette just below the surface of the bromobenzene xylene mixture, with care not to touch the sides of the tube now turn the thumb screw on the pipette holder until the blood is at the level of the next mark, then gently lift the pipette out of the tube, and release the drop of blood. The surface tension existing between the blood and the mixture in the tube causes its release.

7. Time the fall of the drop of blood by pressing the stop watch as the center of the drop passes the upper mark and again as it passes the lower mark, 30 centimeters below on the tube, and record the falling time.

8. Repeat the procedure using another finger if the blood is not flowing freely from the first puncture, and a clean pipette. Two drops of whole blood should not be dropped from the same pipette, for the second drop is always heavier as the result of sedimentation of the cells in the interval during which the first drop is falling. Pipettes should be rinsed with ammonia water immediately after they have been used for whole blood specific gravity, then washed with water, alcohol, and ether. Record the time.

Cleanliness of these capillary tubes is the greatest limiting factor for securing accurate results. Redistilled acetone can be employed to advantage in carrying out this technique.

9. In a similar manner draw up the standard[1] just above the upper mark on the pipette, draw it down even to the mark by touching the tip to a piece of gauze, then determine its falling time between the 2 marks on the tube.

Criteria for checks on falling time. When a drop of blood, plasma, serum, or standard potassium sulphate solution falls in the bromobenzene mixture, successive drops from the same or different pipettes should give apparent density differences of not greater than 1×10^{-4}. It will be observed (Fig. 3) that 0.1 of a second in the rapid range of falling time constitutes a larger range in the apparent density differences than 0.5 of a second difference in the falling time of 2 successive drops at the other end of the scale. When accurate checks can not be made, a sufficient number of drops should be done to establish a good average falling time.

Reasons for inability to get checks on drops. (1) Dirty pipettes cause drops of varying sizes to be delivered. (2) Slipping drop due to rubber tubing in pipette holder being too large causing incomplete suction. (3) Too much tension on thumb screw at moment of release causes rebound of fluid in pipette when drop is released. Thumb screw should compress rubber tubing in holder uniformly and should be released before pipette is removed from tube so that level of fluid in pipette remains at the mark. (4) Torsion of hand steadying tip of pipette increases

[1]*The standards should be preserved under oil;* small portions being removed for daily work.

pressure in rubber tube and, therefore, a rebound suction action occurs at release. Relieve all tension before drop is released. (5) Currents in the dropping tube due to rapidly changing temperature on one side of the water jacket, e.g., that caused by opening a window or lighting a burner near the apparatus. (6) Air bubble or particle of solid material. (7) Poor reaction time in using stop watch. A stop watch stand[1] will aid in accurate timing. (8) Incomplete mixture of the bromobenzene xylene solution causing varying rates of speed in the falling drop. (9) Gross differences in temperature between bromobenzene mixture and plasma. These are minor technical details which are quickly mastered but which in the beginning give trouble.

Determination of the specific gravity. 1. To calculate the apparent density difference between the blood and the mixture through which it falls, insert a pin on the temperature scale (Fig. 3) at the temperature of the water bath and with a thread cross the "falling time" scale at the figure given by the stop watch. The thread will cross the "apparent density difference" line at a point which gives the difference in density.

2. Calculate the apparent density difference between the standard and the bromobenzene xylene mixture in a similar manner.

3. Calculate the true density difference between the blood and the standard by subtracting the lesser figure from the greater.

4. Correct for temperature by subtracting from the specific gravity of the standard 0.0001 for each 2 degrees' change in temperature above 20 degrees C. and by adding 0.0001 for every 2 degrees below 20 degrees C.

5. If the blood falls faster than the standard, it is heavier; therefore, the true density difference is added to the corrected specific gravity of the standard. If the blood falls slower than the standard, it is lighter; therefore, the true density difference is subtracted.

Example:

Specific gravity of standard.	1.0550	
Correction for temperature of 22° C.	−0.0001	
Corrected specific gravity of standard		1.0549
Falling time of blood	21.5 secs.	
Apparent density difference.	0.0115	
Falling time of standard	27.5 secs.	
Apparent density difference.	0.0086	
True density difference 0.0115−0.0086		0.0029
Specific gravity of blood		1.0578

This whole procedure can be carried out in about 2 minutes. This cannot be used to determine proteins. While the proteins do contribute to the specific gravity of whole blood, the red blood cells and their hemoglobin content are more significant because of their greater weight.

Specific gravity of plasma. The steps for determining specific gravity of plasma are exactly the same as those described for whole blood, except that 2 drops are used from each pipette and No. 3 standard (specific gravity 1.0268). The determination is easier because there is no tendency toward coagulation.

For heavy plasma tube P_1 is used (specific gravity 1.0230); for lighter plasma tube P_2 (specific gravity 1.0130). Again the falling time of the plasma is checked against the falling time of the standard and the true density difference is added or subtracted to the corrected specific gravity of the standard.

It is this plasma specific gravity from which the proteins are calculated. It is the plasma specific gravity test which is routinely done on all pre-operative cases; the whole blood specific gravity is determined only in those cases in which difficulty is anticipated or when the course of the patient is being followed at the bedside during emergency treatment.

PROTEINS

In 1927, Atchley and Benedict, after a careful study of the electrolyte distribution in a case of severe intestinal obstruction, suggested that a simple determination of serum protein content might be the best aid in following the degree of dehydration and treatment.

In 1929, Moore and Van Slyke showed that there is a constant relationship between the specific gravity of the serum or plasma and the protein content. For plasma they expressed this relationship by the formula: $P = 343\,(G - 1.0070)$, in which P equals the grams of protein per 100 cubic centimeters of plasma and G equals the specific gravity of the plasma. This work was done on human plasma and the maximum deviation was found to be 0.6 gram per cent.

Weech, Reeves, and Goettsch, in 1936, checked the work of Moore and Van Slyke. In their studies specific gravities were determined by pycnometry and nitrogen determinations by the micro-Kjeldahl method. Their formula for plasma was given as $P = 340.1\,(G - 1.00687) \pm 0.103$. It is this formula which we have routinely used.

We have rechecked Weech's formulas for both serum and plasma using a series of unselected clinical cases, and have found it to be

[1] May be obtained from A. R and J. E. Meyland, New York City

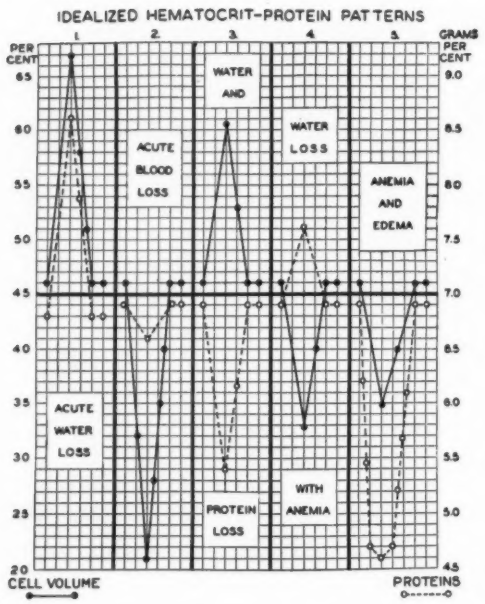

Fig. 4. These patterns represent idealized, superimposed, graphic histories of the changes in cell volumes and plasma protein determinations on several cases of each type with the time element eliminated for the sake of brevity in comparative representations. Each curve begins at a hematocrit reading just above the normal heavy black line; the proteins just below the line and each returns to the beginning position.

Column 1. Acute water loss. Here there is marked hemoconcentration and dehydration as indicated by the rise in both cell volume and protein level. Such a pattern is typical of conditions in which there is excessive vomiting, severe diarrhea, diminished fluid intake, or uncomplicated shock.

Column 2. Acute blood loss. Hemorrhage gives a picture of steadily falling hematocrit reading and moderate protein loss.

Column 3. Water and protein loss. This is the picture so characteristic of severe burns, to a slightly less degree in ruptured peptic ulcers, ruptured appendices with peritonitis, and persistent fistulas from the bowel.

Column 4. Water loss with anemia. Relative hyperproteinemia in the presence of secondary anemia of chronic illness is a fairly reliable index of the degree of dehydration.

Column 5. Anemia and edema. All values are below the normal line. When proteins reach a concentration of less than 5.5 grams per cent functional disturbances related to the water content of the tissue may be expected; when below 5.0, true edema usually supervenes.

very accurate when the protein percentages, as calculated from the specific gravity determinations, were checked by the standard micro-Kjeldahl method for nitrogen determination.[1] Comparing the mean average between the determined and calculated protein values in one series, we found that the difference amounted to ± 0.16 gram per cent or 2.3 per cent.

The essence of the relationship is that plasma, completely free of protein, has a specific gravity of about 1.00687. Only rather large changes in the salt content of the blood upset this constant. The factor of 340.1 indicates that for each increase of 1 gram per cent of protein the specific gravity rises 1/340 or .00294; in other words, each increase in the specific gravity of 0.0001 indicates 0.03 gram per cent increase in protein.

By this method the total protein content values are open to question in several types of cases that have come to light so far. They are: gross hemolysis, severe diabetes, hypercholesterolemia, gross lipemia, and excessive bilirubinemia.

To facilitate the rapid calculation of the total protein content of the plasma from the plasma specific gravity determinations, a chart with comparative values already worked out has proved of assistance. The following is a condensed form of such a chart:

Specific gravity	Protein Grams per cent	Specific gravity	Protein Grams per cent
1.0187	4.01	1.0275	7.01
1.0202	4.52	1.0290	7.52
1.0217	5.03	1.0304	7.99
1.0231	5.51	1.0319	8.50
1.0246	6.02	1.0334	9.01
1.0260	6.50	1.0348	9.49

INTERPRETATION OF STUDY

A fairly definite idea of the history and clinical picture of the patient should always be sought before a final evaluation of the state of hydration is made from the data given by the preceding tests. Most important is the trend toward or away from normal as judged by repeated tests and not a single set of determinations. Certain well defined patterns have recurred many times in following a large series of cases, and these have proved of great aid in the interpretation of the values in any specific case.

[1]In the medical and surgical laboratories through the kindness of Dr. Dana W. Atchley and Dr. Louis Bauman.

In *simple dehydration* (Fig. 4, column 1), whether from lack of fluid intake, diarrhea, excessive sweating, severe vomiting, or shock of a psychogenic, traumatic, or postoperative origin uncomplicated by hemorrhage, there is a rise in the cell volume, the whole blood and plasma specific gravity, and the plasma protein percentage. In the first 3 of these conditions, treatment consists in administering fluids until these elevated values tend to approach normal. In shock, however, the mechanism is more complicated; hence treatment, to be rational, must attempt to overcome first the severe arteriolar and venular spasm, second, the capillary paralysis and dilatation, and third, the great loss of circulating blood volume. Hypertonic sodium chloride is particularly effective in relieving this arteriolar spasm, in decreasing the viscosity of the blood, and in aiding the return of fluid from the tissues into circulation. It should be used cautiously in collapse due to dehydration (26). Suprarenal cortical hormone (eschatin) has proved valuable in restoring capillary tone, raising the blood pressure, and redistributing electrolytes. To maintain any gain initiated by the sodium chloride and eschatin therapy, blood transfusions are given as dictated by the findings on repeated blood studies.

In *hemorrhage* (Fig. 4, column 2), either obvious or concealed, there is an immediate fall in the specific gravity of the whole blood and a drop in the cell volume as determined by the hematocrit. The plasma specific gravity changes are less marked (26). Even in severe hemorrhage, these values may not be abnormal due to readjustments of plasma proteins and circulating volume. They do not approach the percentage loss of cellular elements as indicated by the fall in whole blood specific gravity and the hematocrit.

Treatment here consists of restoring blood volume by transfusions. It is of utmost importance in cases of shock when the patient is unconscious to determine whether that shock is complicated by hemorrhage or not. These simple tests can make this differentiation in the vast majority of cases.

When there is not only loss of fluid but loss of protein as well (Fig. 4, column 3), there is a tendency for the hematocrit curve to rise while the protein values continue to fall. Such patterns are typical of severe burns, ruptured peptic ulcers with peritonitis, and even ruptured appendices with large abscess formation as the result of a great pouring out into the peritoneal cavity of an exudate rich in protein. In these cases, the problem of water balance is doubly hard. The extreme hemoconcentration and shock, if present, must be combated by means of adequate fluid administration; yet, the already lowered protein concentration must not be reduced to the edema level which in most patients is reached at about 5 grams per 100 cubic centimeters of plasma. Ravdin and his colleagues have laid great stress on the part that such hypoproteinemia plays in the malfunctioning of tissues and organs, particularly after traumatic or operative insult.

When acute changes take place in chronic disease (Fig. 4, column 4) in which there already exists an anemia and probably a hypoproteinemia, any one of the ordinary tests, i.e., red cell count, hemoglobin, or hematocrit determination, will not evaluate the true state of hydration. A combination of all these tests is necessary, in particular, the determination of plasma specific gravity. A sudden water loss is reflected by an increase in the weight of plasma before other changes become apparent. This resultant hyperproteinemia is relative. Treatment consists in reducing the protein values to approximately normal levels and then restoring the cellular elements of the blood by appropriate means.

Impending edema (Fig 4, column 5) may be suspected by a gradually falling plasma protein level. Such water logging of tissues is detrimental in any surgical condition. In severe burns local edema causes an early creeping of the tan, allowing infection at its periphery. In postoperative resections or anastomoses, the stomas become swollen and obstructed, the motility of the gut is lessened, and the tendency for ileus increases. A definite relationship exists between the water content of tissues and the healing of wounds, particularly in those cases in which catgut has been used as the suture material and its tensile strength is affected. Repeated determinations of the plasma protein content by an

easy but accurate method assist in the prevention of such complications.

There is no formula at present which will state in a simple manner the amount of fluid necessary to rehydrate any given dehydrated patient, nor is there any rule of thumb for the type of fluid to be used in each case. Certain precautions seem wise; e.g., in cases of hemoconcentration in which the hematocrit reading shows over 60 per cent cell volume, it seems unwise to use large quantities of hypertonic solutions because of the danger of bringing into circulation cell water which is rich in electrolytes definitely toxic when present in the plasma in quantities greater than normal (26).

In the severely ill there may be a complete loss of the ability to utilize fluids of any type in almost any quantities until the severe spasm of the peripheral vessels is relieved and the consequent return to the circulation of the sequestrated blood in the paralyzed peripheral capillaries.

The only safe way at present is to measure the degree of hemoconcentration or anemia, the degree of dehydration or edema, institute the therapy suggested by the findings, follow the curve of progress by repeated determinations, and evaluate the state of hydration day by day or hour by hour if need be.

SUMMARY

1. Four simple tests are presented in some detail which may be used as emergency measures in determining and regulating the state of hydration of acutely ill persons. They are: (1) the determination of the cell volume of venous blood by means of the hematocrit; (2) determination of the specific gravity of the whole blood by the modified Barbour and Hamilton method; (3) determination of the specific gravity of the plasma; (4) calculation of the plasma proteins from the plasma specific gravity by a simple formula.

2. By means of the data thus acquired one may determine degrees of water loss, water plus protein loss, anticipate the onset of shock, differentiate shock due to simple circulatory collapse from shock complicated by hemorrhage, detect dehydration in the presence of anemia, predict the approach of an edema level of proteins, and direct treatment more rationally for the alleviation of any of these conditions.

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THE PROBLEM OF TUBERCULOUS EMPYEMA THORACIS

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TUBERCULOUS empyema is a problem both from the purely academic aspect of classification and from the practical aspect of treatment. The latter has been, and still is, a controversial point. We have been stimulated to review this subject because of the frequency with which we have been faced with this complication and also on account of the difficulties of treatment and the unsatisfactory results obtained.

In the past 30 years the use of pneumothorax has increased greatly and with it the incidence of tuberculous empyema. Among chest specialists there is agreement as to general principles of treatment. Unfortunately, however, the general medical profession, although undoubtedly familiar with these principles, very often fails to apply them. Consequently many of the patients are seen at a stage when operative procedures cannot be carried out. It is generally accepted that prolonged, unsuccessful, palliative treatment leads to a high mortality. In spite of this there is a tendency to keep on with such treatments for months without benefit to the patient. Finally the disease spreads or the resistance is so lowered that more radical measures are impossible.

In this article we have reviewed 37 cases. The mortality has been high, the reasons for which we have attempted to analyze. The progress of the disease has been studied in individual cases in an effort to find out at which stage different treatment would have been more successful. In addition we would like to present a classification of tuberculous empyema based primarily on the etiological factors.

INCIDENCE

The incidence of tuberculous empyema reported by previous authors varies considerably. Reports in recent years have stressed the increasing incidence due to artificial pneumothorax. The following, compiled from statistics given by Alexander (9), shows the frequency with which empyema may occur during the course of pneumothorax treatment: Ulrici, no empyema in 600 pneumothorax cases; Scroder, 2 cases of mild empyema and 1 of severe mixed infection in 300 pneumothorax cases. One-sixth of serous effusions changed into pure tuberculous empyema; L. S. Peters, in 700 pneumothorax cases found pleural effusion in 70 per cent; pure tuberculous empyemas, in 20 per cent; empyema in 7 per cent. Ernest Peters, in 100 pneumothorax cases reports 1 of empyema; Kinsella found empyema in 10 to 12 per cent of his pneumothorax cases; Hayes, in 21 per cent of 151 pneumothorax cases; Jones and Alexander, of 70 empyemas, 28 complicated pneumothorax.

Our own findings are shown in Table I. We would like to point out that in our own series there is a total incidence of 2.2 per cent. If, however, the cases of artificial pneumothorax are not included, the incidence of empyema is then reduced to 0.5 per cent in a total of 1,371 cases. If only pneumothorax cases are considered, the incidence is 9.9 per cent. That is, once a pneumothorax is induced, the chances of getting an empyema are 20 times greater than if the patient were treated by other methods.

LITERATURE

In reviewing the literature on tuberculous empyema from 1915 to January, 1939, we have been impressed by the relatively few comprehensive studies which have been made and, what is more, by the comparatively few cases presented in the best studies. Prior to 1919 there appeared in the literature occasional references to tuberculous empyema, usually in the form of a case report. In this year Duboff reported a series of 20 cases of tuberculous empyema which occurred among

From the Grace Dart Home Hospital.

TABLE I.—PRESENT SERIES

	Number	Empyemas	Percentage of empyemas	Dead or dying—per cent	Stationary or well—per cent
Total admissions	1,675	37	2.2	72.9	27.1
Total induced pneumothoraces	304	30	9.9	73.3	26.7
Total spontaneous pneumothoraces	22	7	31.8	71.4	28.6

Note: 81 per cent of empyemas were secondary to induced pneumothoraces.

902 patients. Of these, 48 had had artificial pneumothorax. Ten of the empyema cases occurred among those cases receiving artificial pneumothorax treatment. Duboff recognized the fact that induced pneumothorax was the most potent cause of tuberculous empyema and this is usually accompanied by extension of lung disease with rupture of the lung into the pleural space. In the treatment of his cases he aspirated for pressure symptoms and remarked, "None of our cases have been cured in the sense that the pleural space has been cleared of pus and has healed without persistent sinus and we have used all methods except thoracoplasty."

Strangely enough, 20 years later, it is apparent from our own study that artificial pneumothorax is the greatest single cause of tuberculous empyema and, what is more, the underlying etiological factor is that of lung rupture. The importance of a bronchopleural fistula apparently was recognized by Duboff and more recently has been confirmed by Coryllos.

Peters in 1921 reported 26 cases of purulent effusions occurring among 250 cases of artificial pneumothorax. Eight of these were cases of mixed infection and 18 were of the pure tuberculous type. Thoracotomy drainage was done on all the mixed infection empyemas. Six of the 8 died, 1 had thoracoplasty and was alive but not well, and 1 recovered. Of the pure empyema cases 10 were followed, 4 carried pus for 3 years in reasonable health, 2 were still under treatment, and 4 were dead. Peters recognized that the mixed infection empyemas were due to a superimposed spontaneous pneumothorax but did not come to the same conclusion about the pure tuberculous cases. In the treatment of the pure cases, aspiration was used to alleviate pressure symptoms.

Jehn, in 1921, in a non-statistical article drew attention to important facts which are fairly well accepted today. He cautioned against thoracotomy except in severe mixed infection, suggesting repeated aspirations with gas replacement (nitrogen), and advocated thoracoplasty soon after thoracotomy where the latter is done. He stated that thoracoplasty was especially indicated in cases in which pneumothorax and pleural exudate cause only partial collapse of the lung with a remaining open cavity in the uncollapsed portion. He also stressed the need to aspirate between thoracoplasty stages to obtain best results.

McKinnie, in 1922, stated that 5 per cent of serous effusions complicating artificial pneumothorax became purulent and not infrequently were converted into the spontaneous type of pyopneumothorax. He reported 28 cases of mixed tuberculous empyema which were treated by aspiration and air replacement, and by open thoracotomy drainage, with poor results.

Hedblom (6), in the same year, reported a series of 76 cases. He pointed out that aspiration of pure tuberculous empyema should not be carried out indefinitely because of the danger of mixed infection and of marked progressive thickening of the pleura. In such cases he advocated thoracoplasty. In the presence of mixed infection Hedblom advised prompt drainage, open or closed, with or without irrigation.

Mainin, in 1926, reported a series of 359 cases of pulmonary tuberculosis in which he was able to induce pneumothorax in 205 cases. In patients thus treated 46 per cent developed a serofibrinous effusion, 7.8 per cent an opalescent effusion, 5.8 per cent empyema, and 2.4 per cent empyema with fistula. He used aspiration, irrigation, and air replacement, and reported improvement but did not give his results in detail.

Archibald, in 1930, presented a classification of tuberculous empyema based on the type of fluid present in the pleural space. He divided his cases into three types: Type I,

TABLE II.—RESULTS OF VARIOUS TYPES OF TREATMENT—246 CASES

	Well per cent	Worse per cent	Dead per cent
Simple aspiration	30	30	40
Closed drainage	29	21	50
Open drainage	32*	6	56
Thoracoplasty	55	13	30

*15 per cent improving.

seropurulent effusion with straw colored turbid fluid containing tubercle bacilli; type II, purulent effusion positive to guinea pig; type III, purulent effusion with many contaminating organisms. Archibald reported a series of 34 cases treated by thoracoplasty: type I, 8 cases; type II, 11 cases; and type III, 15 cases. In type I there were 3 practical cures and 5 cases were improved. In type II there were 4 improved and 2 practical cures. In type III he reported 7 on whom thoracoplasty was done, with 2 improved. In 8 cases simple thoracotomy was done but further operation was contra-indicated and all did poorly.

The Empyema Committee Report (5), read before the American Sanatorium Association in 1931, was based on the replies of 56 surgeons from both continents concerning their method of treatment of tuberculous empyema. Nearly 50 per cent of the surgeons resorted to open or closed drainage in cases of type I and type II infection (Archibald). Table II indicates the results reported for various types of treatment in 246 cases.

The Committee's report, while interesting, failed to mention the number of bilateral cases. Any data which do not take this into account must of necessity leave room for great variations in the end-result. The Committee pointed out that "too much time must not be given to the so called palliative methods of treatment but that radical surgery should be advised early if the condition of the patient warrants the operative risk."

Carl Hedblom (7), in 1932, reported 143 cases of bacteriologically proved tuberculous empyema. He contrasted the results of palliative and radical surgical measures. In cases of tuberculous empyema without pyogenic infection, thoracoplasty cured 74.7 per cent in a group of 23 patients without any mortality. In the cases with pyogenic infection 64.4 per cent were cured or markedly improved and there was a 20 per cent mortality in a group of 59 cases. As a comparison those patients treated by palliative measures showed 21.4 per cent cured and a mortality of 32.1 per cent, in a group of 28 cases of pure tuberculous empyema. Of the mixed infection tuberculous empyemas 12.1 per cent were cured, and there was a mortality of 42.1 per cent in a group of 33 cases. Hedblom concludes that "eventually most patients with unobliterated tuberculous empyema cavities die of the condition."

Jones and Alexander, in 1934, presented a study of 70 consecutive cases of tuberculous empyema, in which they followed Hedblom's classification. In 43 of the 70 cases, thoracoplasty was resorted to, with the result that 74.4 per cent of the patients were considered cured, 7 per cent were improved, and there was a mortality of 7 per cent.

In the same year Rosenblatt reported a series of 21 cases of toxic tuberculous empyema treated by aspiration, air replacement, and the injection of 2 or 3 cubic centimeters of saturated alcohol solution of methylene blue. Rosenblatt reported 52 per cent cured and 48 per cent dead. Rosenblatt failed to classify his cases, nor did he say whether he was dealing with unilateral or bilateral disease.

MacDonald reported in 1936 that he divided his tuberculous empyema cases into two groups: simple tuberculous and tuberculous with mixed infection. In the treatment of simple tuberculous empyema he favored aspiration and air replacement. In those cases of mixed infection in which the infection was mild he employed the same procedure. He resorted to drainage in those cases of mixed infection which were toxic. A total of 15 cases of mixed tuberculous empyema was presented. Eleven of these were severe and 3 mild. Of the severe cases 5 had extrapleural thoracoplasty, and 6 "undecking" operations. Thirteen of his cases were then living, 10 were well and working, 2 were improved, and there was 1 death.

Leaver and Hardaway, in 1937, presented a total of 59 cases, 35 of which were pure tuberculous empyemas and 24 of which were mixed. They reported that 5.8 per cent of

750 artificial pneumothorax cases developed tuberculous empyema. Sixty per cent of these tuberculous empyemas were attributable to spontaneous pneumothorax superimposed on the artificial pneumothorax, while 40 per cent developed from clear fluid. Of a total of 35 cases of pure tuberculous empyema, 57.1 per cent were cured, 20 per cent were improved or still under treatment, and 22.9 per cent were dead. Of these cases 51.4 per cent had oleothorax alone, and 5.7 per cent of the cured cases had thoracoplasty in addition to oleothorax. Of the 24 cases complicated by mixed tuberculous infection 20.8 per cent were cured, 8.3 per cent were living but worse, and 70.9 per cent were dead. All cured patients required complete thoracoplasty.

Coryllos, in the same year, drew attention to the high frequency of bronchopleural fistulas in tuberculous empyema. By means of gas analysis of the pleural air Coryllos was able to demonstrate the presence and estimate the size of a bronchopleural fistula. He divided fistulas into 3 types: (1) punctiform fistulas; (2) fistulas of moderate size, and (3) large fistulas. He stated that "in pulmonary tuberculosis the production of purulent exudate in the pleural cavity or the change of existing clear effusion to purulent ones is always due to the development of pleuropulmonary fistula." He believed that in those cases in which the empyema is carried for months or years the fistula becomes sealed off, otherwise the pus would have been coughed up.

Woodruff, in 1938, reported a series of 147 cases, which he divided into pure tuberculous empyemas (group A) and tuberculous empyemas with mixed infection (group B). In group A there were 105 patients, of whom 28 per cent were dead, 29.9 per cent were resting, and 42 per cent were well and working. In group B there were 42 patients, of whom 59.5 per cent were dead, 23.8 per cent were resting, and 16.6 per cent were well and working. In this group of cases some patients were treated by irrigation with salts and dyes, some by oleothorax, and others by thoracoplasty. He concluded that thoracoplasty was of great value in groups A and B, the other measures being of some value in group A only. In conclusion Woodruff stated:

"Many of the dead who have not had thoracoplasty probably passed through a stage where thoracoplasty could have been done with minimal risk."

Penington, in 1939, concluded an article on tuberculous empyema with the following statement:

"In a small series of cases of tuberculous empyema an easily demonstrable rupture of a caseating focus in the lung was invariably found at autopsy.

"In an experimental study of the production of tuberculous pleural effusions in the rabbit it was found to be impossible to produce an empyema by simple inoculation of the pleural sac with tubercle bacilli even in a sensitized animal. Nor did subsequent trauma or pleural inoculation of whole blood lead to empyema. An empyema was obtained experimentally only when a caseating tuberculous focus extended from the lung to the pleura and ruptured. Apparently the presence of caseous material in a free pleural space is the necessary condition for the development of an empyema.

"Experimental and pathological evidence is thus in accord and supports the belief that tuberculous empyema in man is the result of the rupture of a caseating tuberculous focus through the pleura."

ETIOLOGY

The pleura can be infected in the following ways: (a) bronchopleural fistula; (b) direct extension from tuberculous pneumonic or bronchopneumonic disease; (c) hematogenous, lymphatic, and other rare sources, such as, needle puncture, tuberculosis of ribs and glands.

a. In the treatment of pulmonary tuberculosis there are certain complications which greatly alter the prognosis. Perhaps the complication most to be feared is that of bronchopleural fistula. When this occurs there is a pathway directly from the mouth to the pleural space, along which mouth organisms can travel. The degree of the pleural infection will depend on the size of the fistula. Clinical experience has shown that a large bronchopleural fistula often causes a dangerous mixed infection, whereas a small fistula may or may not be responsible for secondary infection of the pleural space. There are three common causes for a bronchopleural fistula in tuberculous lungs, namely: (1) tear of the lung due to the pull of an adhesion, (2) rupture of a small subpleural caseous focus, and (3) rupture of a large cavity.

When these causes are borne in mind, the presence of bronchopleural fistula in tuberculous empyema is understandable. The frequency and importance of bronchopleural fistula in tuberculous empyema was not fully appreciated until recently. Coryllos, by means of gasometric analysis of the pleural air, was able to demonstrate bronchopleural fistula in all cases of tuberculous empyema. Further, the same author showed that there was a definite relationship between the size of a fistula and the degree of pleural infection. From this it would appear that the development of a tuberculous empyema signifies the presence of a bronchopleural fistula. It is reasonable to assume that the longer the fistula remains, the greater are the chances of mixed pleural infection. Thus pure tuberculous empyemas frequently progress to the virulent mixed type of empyema. In our concept tuberculous empyema, like tuberculosis elsewhere, is not a static disease but one which is constantly changing. The pleural fluid which starts as a turbid yellow liquid containing acid-fast organisms may progress to thick greenish pus containing the same organisms. Later this purely acid-fast empyema may become contaminated with other organisms. The time interval between the various phases varies in different patients.

In the section on "Incidence" it may be seen that, since the advent of artificial pneumothorax treatment, tuberculous empyema occurs more frequently than in the past. Coryllos has also shown that bronchopleural fistula occurs during the course of artificial pneumothorax in a high percentage of cases. The frequent and often undetected presence of bronchopleural fistula in these pneumothoraces accounts for the increase in tuberculous empyema which has occurred in the past 25 years.

b. A tuberculous pneumonic or bronchopneumonic disease is always associated with some infection of the visceral pleura. If the visceral and parietal pleural surfaces are in contact, this may result only in adhesions. However, when there is a large amount of exudation before adhesions are formed, the exudate collects in the pleural cavity as an empyema. If the pleural surfaces are separated, as in artificial pneumothorax, the exudate collects at the bottom of the pleural cavity, forming a pyopneumothorax. Once again it is evident that the presence of pneumothorax predisposes to the formation of tuberculous empyema.

c. Tuberculous empyemas developing from hematogenous and other causes are comparatively rare.

DIAGNOSIS

Diagnosis depends on tubercle bacilli in the aspirated fluid and the proper examination of the aspirated material. It is not absolutely necessary, however, to find tubercular organisms in order to diagnose tuberculous empyema. A patient having pus in the pleural cavity, even though it is apparently sterile, must be considered tuberculous if active disease is present in the lung. A certain percentage of empyemas fail to show organisms on examination of the pus, although it is likely that many of these would be found positive if the more refined methods of examination were used.

In the past year at the Grace Dart Home Hospital we have cultured all effusions, using the method of Guernon, and have found it to be most satisfactory. In many cases in which ordinary methods (smear and concentration) have failed to demonstrate acid-fast organisms, we have been able to do so by culture. By this method positive results may be obtained within 4 or 5 days. As compared with guinea-pig inoculation, the period of diagnosis is materially shortened. Guinea-pig inoculation, however, should not be neglected, and all fluid should also be cultured for pyogenic organisms.

An appreciable amount of fluid in the pleural cavity should be regarded with suspicion, especially if it persists for several weeks. The physical and roentgenological signs are too well known for repetition here. We would like, however, to draw attention to certain serious types of cases.

a. Spontaneous Pneumothorax

H.McM., No. 3230, male, aged 40 years. Symptoms first noticed in January, 1938, with cough, night sweats, loss of weight and dyspnea. When pa-

tient was admitted to the Grace Dart Home Hospital there was found a left-sided empyema with a bronchopleural fistula and spontaneous pneumothorax. A moderate amount of disease was present in the right lung. Culture of the empyema fluid revealed acid fast organisms and Streptococcus anhæmolyticus. Despite frequent aspirations of 1,100 to 1,300 cubic centimeters once or twice a week, the clinical course was downhill. Patient lost weight rapidly, pulse was rapid and temperature high. Two months after admission, thoracotomy drainage was performed. The patient died 2 weeks after this operation.

This case illustrates the rapidity with which mixed tuberculous empyema may progress. Conservative measures of drainage, aspiration, and thoracotomy were ineffective. As the disease already affected both sides when patient entered the hospital, no other measures could be instituted in this particular case.

b. Artificial Pneumothorax

G.B., No. 2874, male, aged 58 years. Onset of symptoms occurred in December, 1932. In January, 1934, artificial pneumothorax was induced on the right side. This pneumothorax was incomplete and inefficient; it failed to convert the sputum on account of an adherent upper lobe. Fluid was first observed on October 31, 1935, at which time pneumothorax was discontinued, but the air failed to absorb. In August, 1936, this patient, who was in reasonably good health up to this date, was suddenly seized with a paroxysm of coughing. He immediately raised from 4 to 6 ounces of fluid sputum and became increasingly dyspneic. After this his cough became worse and at times he would suddenly cough up large quantities of purulent watery sputum. He was admitted to the Grace Dart Home Hospital in a moribund condition on August 28, 1936, and died 1 week later.

This case illustrates the danger of continuing an incomplete pneumothorax and also the rapidity of downhill progress in the presence of mixed tuberculous empyema. It is very probable that in August, 1936, the severe paroxysm of coughing was associated with a ruptured adhesion, with consequent bronchopleural fistula and mixed infection.

C.G., No. 3025, male, aged 40 years. Onset of symptoms occurred in 1932. In January, 1933, a left sided artificial pneumothorax was commenced. After pneumothorax was induced, the patient did well and was an ambulatory case with negative sputum until May, 1937. At this time he experienced severe pain in the left chest, accompanied by a sudden rise of temperature to 103 degrees. Within a short time purulent fluid formed in the left pleural cavity. Prior to May, 1937, the right lung was healthy. Within a month there was evidence of a marked bronchogenic spread to the right base. A review of the roentgenograms showed that a cavity at the left apex had ruptured prior to the formation of the empyema. The patient died August 13, 1937, and the clinical findings were confirmed by autopsy.

Although this patient had been doing well clinically for nearly 4 years, the pneumothorax had not collapsed the cavity. Later, rupture of the cavity resulted in bronchopleural fistula, bronchogenic spread, tuberculous empyema, and early death (Fig. 1).

PROPHYLAXIS

As we have previously stated, the most frequent cause of tuberculous empyema is bronchopleural fistula. By following simple common sense measures it should be possible to curtail the incidence of fistulas. In cases of active pulmonary tuberculosis the most important preventive measure is the avoidance of effort. Activities which cause sudden changes in intrapleural pressures are especially dangerous. This is more particularly true of cases of artificial pneumothorax which are complicated by adhesions. A sudden cough or straining effort may result in the rupture of an adhesion. Many workers attempt to rupture adhesions by increasing the intrapleural pressures, thus hoping to improve artificial pneumothorax collapse. Such adhesions may contain lung tissue or may rupture at their pulmonary attachments, thus creating bronchopleural fistulas. The relationship between bronchopleural fistula and tuberculous empyema has been mentioned several times in this article.

We are of the opinion that adhesions under tension should be cut when possible. On account of technical difficulties, pneumonolyses cannot always be executed. In such cases, if pneumothorax is not efficient, it should be abandoned and replaced by surgical collapse. If, however, the pneumothorax is efficient, it may be continued but the activity of the patient should be drastically curtailed. Brown, in 1936, pointed out the relatively high incidence of inefficient pneumothorax with its attendant danger. In our own series the ma-

jority of tuberculous empyemas developed in cases in which pneumothorax therapy was continued long after it should have been abandoned. The continuance of an inefficient pneumothorax may result in one or both of two complications: (1) spread of the disease from an uncollapsed cavity, (2) tuberculous empyema secondary to ruptured adhesions and the resultant bronchopleural fistula (see Case of C. G., here reported above).

CLASSIFICATION

I. Tuberculous empyema with bronchopleural fistula:
   a. Complicating artificial pneumothorax
      1. Pure,
      2. Mixed
   b. Complicating spontaneous pneumothorax—
      1. Pure,
      2. Mixed
II. Tuberculous empyema without bronchopleural fistula.
III. Same as type I or type II, with bilateral active parenchymal disease.
IV. Idiopathic—
      1. Pure,
      2. Mixed

The significance of bronchopleural fistula in relation to tuberculous empyema has been accepted for some time on purely clinical grounds. More recently, however, as we have previously mentioned, Coryllos has demonstrated the frequency and importance of bronchopleural fistula in tuberculous empyema. Coryllos, using gasometric analyses of artificial pneumothorax cases, was able to show that spontaneous pneumothorax occurred frequently during the course of artificial pneumothorax treatment. According to this author, practically all cases of tuberculous empyema had a small or large bronchopleural fistula.

The classification presented here attempts to take into consideration this etiological factor and at the same time include others essential for treatment. Previous classifications of tuberculous empyemas have not completely taken into account the underlying etiological factors.

RESULTS OF CASES

In our series there was a total of 37 cases of tuberculous empyema. We wish to present brief summaries of some of these cases. Each case presented illustrates a different type of empyema indicated in the classification given.

TABLE III.—TYPES OF EMPYEMA

	Number	Unilateral	Bilateral	Unilateral turning bilateral
Pure tuberculous empyema	26	14	19	7
Mixed tuberculous empyema	11	5	8	2
Total	37	19	27	9
Per cent		51.3	48.7	

Note: 9 of 19 unilateral cases, or 47.4 per cent, became bilateral.

TUBERCULOUS EMPYEMA WITH BRONCHOPLEURAL FISTULA

Unilateral pure tuberculous empyema complicating artificial pneumothorax. In this group there were 13 cases. The duration of the pneumothorax to the time of onset of fluid ranged from 9 days to 65 months. The time interval from the first appearance of fluid to the onset of empyema varied from 2 weeks to 14 months. The period during which surgery might have been done with benefit to the patient varied from 1 month to 2 years. In the majority of cases, however, it was about 6 months. Although surgical intervention is indicated as early as possible in this group of patients, the urgency is not as great as in the mixed empyema types as will be shown. Under conservative treatment several of these cases became bilateral and consequently inoperable. Six of the patients have died, 3 are going downhill, and in 3 the course of the disease has been arrested. One case is stationary. Of the arrested cases 2 underwent thoracoplasty and one was cleared up by aspiration and air replacement.

Two of the 7 cases which became bilateral did so shortly after thoracotomy. The dangers of thoracotomy will be discussed later under "Treatment."

J.N., No. 2770, male, aged 26 years. Symptoms of disease commenced 3 months prior to admission to the Grace Dart Home Hospital. One month after onset, a left pneumothorax was induced. The patient progressed favorably until 3 days prior to admission, at which time he developed a spontaneous pneumothorax superimposed upon the artificial. Fluid began immediately to accumulate in the pleural cavity. At first it was straw colored, then slightly turbid, and became frankly purulent 5 months later. No pyogenic bacteria were found by culture but many acid fast organisms were demonstrated. Thoracoplasty was commenced 1 month

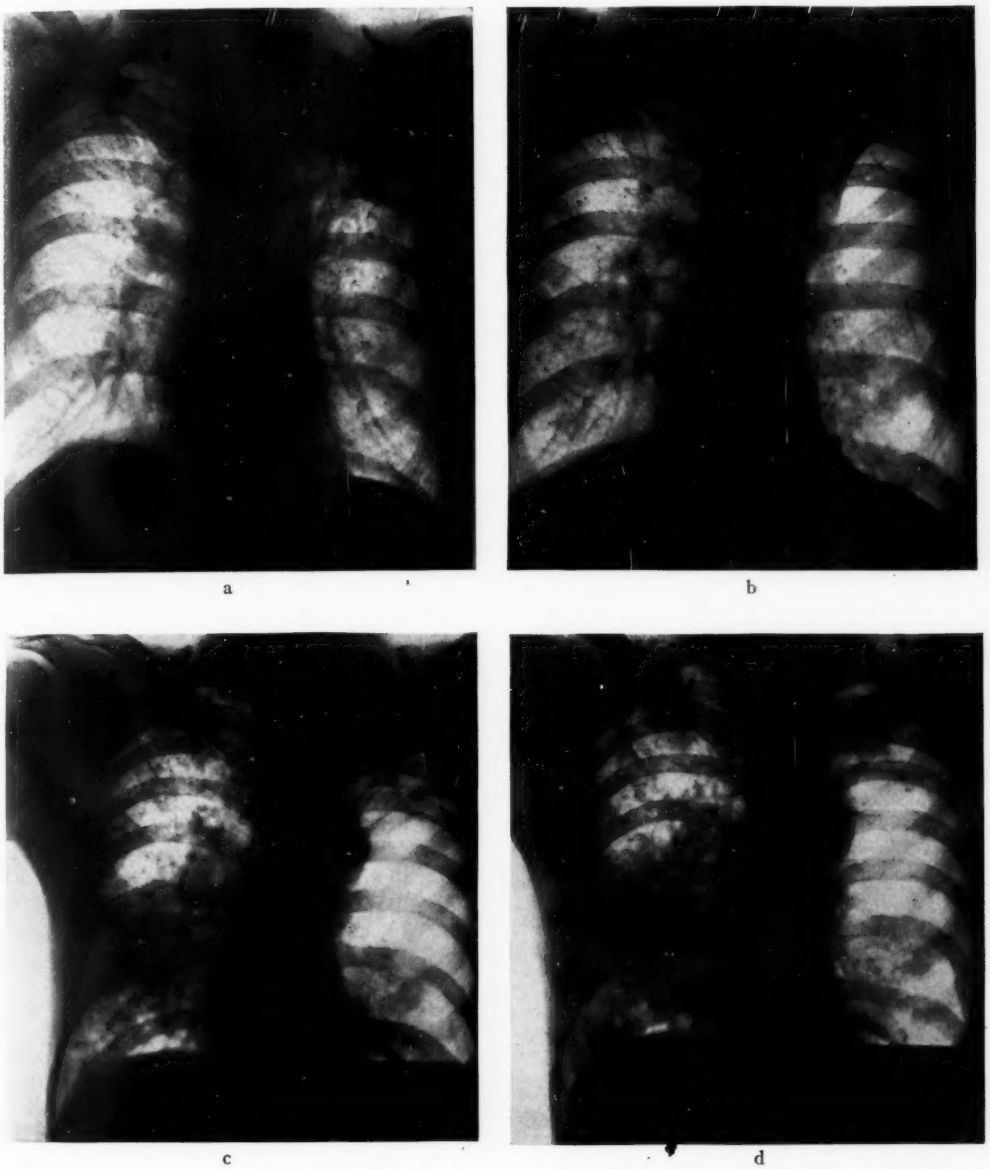

Fig. 1. a, May 23, 1934. The left upper lobe is involved in a tuberculous lesion showing a large cavity. The right lung is comparatively clear. b, May 4, 1937. The pneumothorax on the left has collapsed practically all of the left lung *except the cavity at the apex*. Because the patient's sputum was reported negative by direct smear the pneumothorax had been kept up. c, June 30, 1937. The cavity in the left upper lobe has ruptured and there is now a tuberculous hydropneumothorax on the left and an extensive tuberculous spread in the right lower lobe. d, July 13, 1937. Shows continued spread of the lesion prior to death. This case illustrates the dangers of allowing a contra-selective collapse to be continued even if the patient is apparently doing well.

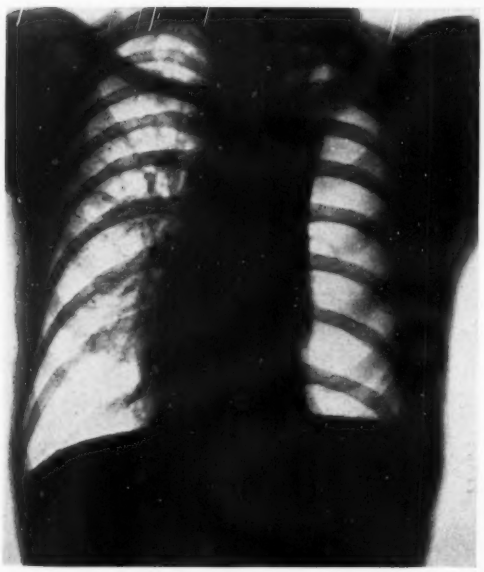

Fig. 2. a, left. March 20, 1936. On the left side there is a tuberculous hydropneumothorax with complete collapse of the lung except for some adhesion of the upper lobe. The right side is clear. b, February 1, 1938. The result obtained by thoracoplasty supplemented by a phrenic operation. There is a large high stomach bubble in this plate and the level of the diaphragm is indicated by an arrow. The patient is sputum free and working part time.

after the empyema appeared. Four stages of operation in all were performed, with good but not complete obliteration of the empyema cavity. There was a decrease in the amount of sputum, which did not turn negative until the thoracoplasty was finally supplemented by a phrenic operation. The small residual empyema cavity healed after thoracotomy. The patient is now working part time as a taxicab driver 1½ years after his last operation and is sputum free (Fig. 2).

M.K., No. 2872, male, aged 56 years. Symptoms of disease commenced 5 months prior to admission. One month before admission a right pneumothorax was instituted. Fluid formed within a month and at first was negative to acid fast organisms but became positive within 2 weeks. At no time were pyogenic organisms demonstrable. By means of repeated aspirations and air replacement the pleural space was gradually obliterated. The sputum, however, continued to be positive. This patient refused thoracoplasty and his condition is at present stationary, with a healed empyema but an open pulmonary lesion.

Unilateral mixed tuberculous empyema complicating artificial pneumothorax. In our series there were 4 cases of unilateral disease with mixed tuberculous empyema which developed during the course of artificial pneumothorax treatment. The duration of the pneumothorax up to the time of onset of fluid ranged from 4 months to 48 months. The time interval between the onset of fluid and the development of empyema was comparatively short. In all of these cases thoracoplasty could have been performed at the onset. As we have stated previously the development of a mixed tuberculous empyema must be considered as a surgical emergency. In only 1 case did the period of operability extend to as long as 3 months. All the patients died within a short time of the onset of the empyema—1 actually within 5 weeks. Two of the 4 cases became bilateral; 1 patient had a thoracotomy and phrenic exeresis and survived for 1 year.

W.H., No. 2580, male, aged 28 years. Onset of symptoms occurred 2 years prior to admission. Pneumothorax commenced on right side 6 months after onset. This was incomplete, with the apex adherent. Approximately 8 months later fluid developed, and it became purulent within 2 months. The sputum was positive and copious. Phrenic

exeresis was performed 2 months after the empyema had developed. Thoracotomy was performed 4 months later. The disease became bilateral and the patient died. There was a period of 3 months after the development of the empyema during which the patient's condition would have withstood thoracoplasty.

Unilateral tuberculous empyema complicating spontaneous pneumothorax. Of this type there were only 2 cases—one pure and the other mixed. In both, fluid developed within a few weeks after the onset of pneumothorax and rapidly became purulent. On both thoracoplasty was performed. In the case of the mixed infection it was done early and was preceded by thoracotomy. In both patients the disease is clinically arrested, but one has a persistent sinus.

TUBERCULOUS EMPYEMA WITH BILATERAL DISEASE

There were 27 cases with bilateral disease. Nine of these had been unilateral when first seen. Nineteen of the cases had pure, and 8 had mixed, empyemas. Twenty-two occurred during the course of artificial pneumothorax and 5 resulted from spontaneous pneumothorax. There have been 17 deaths, 7 are going downhill, in 2 the disease has apparently been arrested, and 1 case has not been traced. Both of the arrested cases underwent thoracoplasty; 1 of these was a mixed, and the other a pure, tuberculous empyema.

In an analysis of our cases (Table IV) we have been struck by the high mortality as compared with reports presented in the literature. This we believe is due to the fact that we have considered all cases instead of selecting only those suitable for therapy. A consideration of the patients who did well reveals the significant fact that 5 of a total of 6 cured cases had undergone thoracoplasty. After analyzing our own cases individually we are convinced that many would have been benefited by thoracoplasty had this procedure been instituted sooner.

TABLE IV.—ANALYSIS OF CASES

	Number of cases	Cured or arrested	Stationary or improved	Worse	Dead
Unilateral pure	14	4	2 improved	2	6
Unilateral mixed	5	1	..	..	4
Bilateral pure	12	..	1 stationary	4	7
Bilateral mixed	6	1	1 stationary	..	4
Total	37	6	4	6*	21*

*Not improved 72.9 per cent.

TREATMENT

The development of tuberculous empyema must be considered as one of the gravest complications of pulmonary tuberculosis. Early recognition of the empyema is essential. It is one of the few complications in which well timed surgical intervention may be a life saving measure. Certainly it is evident from the foregoing data that failure to intervene results in a high mortality. Once an empyema is diagnosed, the problem is no longer a purely medical one. The skilled cooperation and constant observation of a medicosurgical team is essential, so that at any stage in the development of the disease, if medical treatment seems inadequate, surgical measures may be rapidly instituted before the patient becomes a poor operative risk. Many of our patients show the rapidity with which serous fluid may progress to the empyema stage and, what is more important, the speed with which patients lose ground and pass from a condition of unilateral operable disease to one of bilateral inoperable disease.

Before deciding on the treatment of a case of tuberculous empyema, one must attempt to classify it as to the underlying etiological factors and as to the type of pus in the pleural space. In all cases of pulmonary tuberculosis in which pleural fluid is suspected the patient should undergo aspiration of the pleural cavity. The fluid should be examined, and, if it persists, should be re-examined at intervals of 3 weeks or oftener. It is important to remember that fluid in the pleural space is not a clinical entity but that there is usually progressive, active underlying disease. This disease is not static but is constantly changing, and at any time a ruptured subpleural focus or lung cavity may result in a small or large bronchopleural fistula. Such a complication changes the clinical picture. Treatment, therefore, must take into consideration not only the type of pus present in the pleural space but also the condition of the underlying lung.

Fig. 3. a, left. March 8, 1938. The left side shows a tuberculous hydropneumothorax. The pneumothorax is inefficient and the cavities in the lung remain open. The lesions in the right lung appear hard and fibrotic. b, April 28, 1938.

Following thoracotomy the left lung has expanded somewhat resulting in a spread to the right side which now shows active exudative disease most marked in the lower hilar region and right lower lobe.

Aspiration with air replacement. There is a small group of patients who respond to aspiration and air replacement or pleural irrigations alone. The majority do not and will require thoracoplasty with or without thoracotomy. We feel that pure empyemas, developing secondarily to artificial pneumothorax with unilateral disease, should be given a trial with aspiration and air replacement. The duration of this treatment should not be prolonged unless there is progressive diminution of accumulated fluid between aspirations. It may require 2 or 3 months of repeated aspirations in order to determine whether the empyema is improving or not. However, one should not persist in this form of treatment even as long as 2 months if the patient is clinically going downhill, inasmuch as thoracoplasty may be needed and should be started while the patient is in good condition.

In cases of bilateral disease with pure empyema, in which thoracoplasty is contra-indicated, aspiration and air replacement may be of some value and should be continued. In those cases in which fluid accumulates rapidly, requiring 2 or 3 weekly aspirations of 400 or 500 cubic centimeters, there is either an extensive pleural infection or a large bronchopleural fistula. In this group aspirations should be abandoned within 2 weeks or less and thoracoplasty started immediately. It will be necessary to continue the aspirations between thoracoplasty stages. Here, however, aspiration is performed alone without air replacement.

Irrigations. Generally speaking irrigations are of the greatest value in those cases with thick pus containing fibrin clots. For the first two or three irrigations it is well to use saline, followed by an instillation of methylene blue for diagnostic purposes. In the absence of a large bronchopleural fistula other irrigating fluids may be used. At the Grace Dart Home Hospital Dakin's fluid has been the irrigating fluid of choice.

Surgical procedures. Some of our cases illustrate that, if surgery had been undertaken earlier, there would have been a greater chance

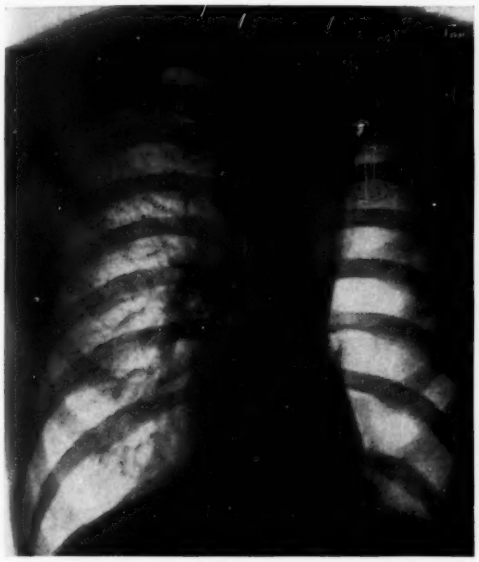

Fig. 4a

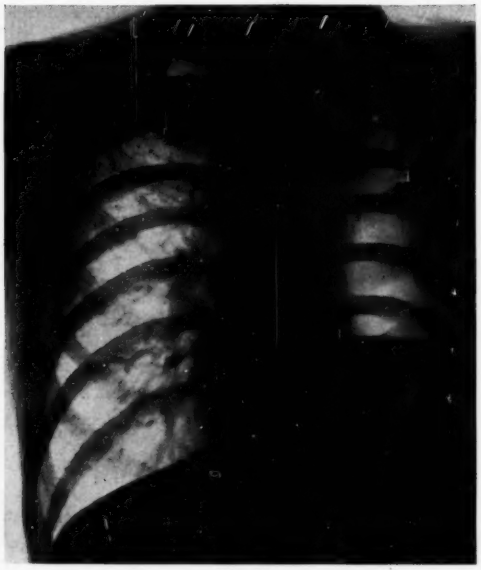

Fig. 4b

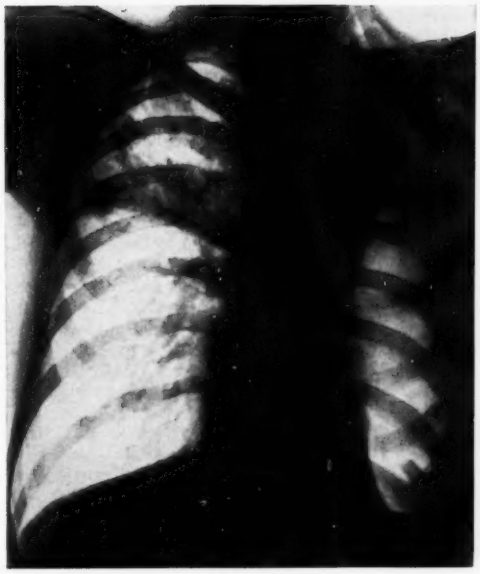

Fig. 4c

Fig. 4. a, January 30, 1936. The left side shows a pneumothorax with complete collapse except for an adherent apex. The right upper lobe shows signs of old disease but is comparatively clear. b, May 12, 1937. A tuberculous hydropneumothorax is present on the left side. The lung is collapsed except for the apex. The right side is unchanged. c, July 28, 1937. Following thoracotomy the cavity in the uncollapsed left apex opened widely and there was a spread to the right upper lobe. Thoracoplasty was started in an attempt to close the cavity but was abandoned after the first stage. This plate taken after the thoracoplasty shows the thoracotomy tube in place and the marked spread on the right side. This case illustrates two dangers: (1) that of continuing a pneumothorax with an adherent apex; (2) that of doing a thoracotomy before the apex is collapsed by thoracoplasty, thus running the risk of opening the cavity in the upper lobe with danger of consequent spread to the other side.

of survival. As we have stated above, pulmonary tuberculosis is not a static disease. There is an optimum period during which thoracoplasty can be performed for treatment of tuberculous empyema. In many cases the period is very short.

In the surgical treatment of tuberculous empyema there are two main objectives: the first is the obliteration of the infected pleural space; the second is the maintenance of collapse of the diseased lung. Both of these can be accomplished by thoracoplasty. It is well known that thoracoplasty, as it is done today, *per se* carries a relatively low mortality rate. For this reason it is necessary to subject patients with tuberculous empyema to thoracoplasty in the early stages of the disease

before there is a spread of the tuberculous process and before the patient has lost his resistance. In our own series many of the patients came to operation late for various reasons beyond our control. In spite of this, thoracoplasty was carried out with good results in a few cases. It is the only procedure which offers the tuberculous empyema patient any real hope of survival.

In cases of pure empyema thoracotomy should under no circumstances be performed. We have seen patients with pure tuberculous empyema drained by thoracotomy and thereby develop severe mixed tuberculous empyema resulting in early death. The opening of the pleural space not only encourages secondary infection but results in re-expansion of the diseased lung. Previously closed cavities reopen and infected sputum accumulates. Very often this material spreads through the bronchial tree and infects the contralateral lung. We have seen 3 such cases, of which the following is an example.

J.G., No. 2932, male, aged 31 years. Onset of pulmonary tuberculosis occurred in 1934. The patient was admitted to the Grace Dart Home Hospital, January 12, 1937, with bilateral disease and left-sided cavitation. In April, 1937, a left pneumothorax was commenced. It was incomplete and did not close the cavity. The sputum remained positive, although the right side cleared almost completely. On December 15, 1937, clear yellow fluid was aspirated from the left thorax. The fluid was frankly purulent by February, 1938, but failed to show organisms on smear. Roentgen-ray and physical examination carried out on March 8, 1938, showed that the right lung was continuing to improve. On March 26, 1938, thoracotomy under local anesthesia was performed on the left side, following which there was a spread of the disease to the right or contralateral base. The spread is well shown in the x-ray plate of April 28, 1938 (Fig. 3). The patient died May 27, 1938, 2 months after thoracotomy.

In the treatment of mixed tuberculous empyema one is faced with a more acute problem. A secondarily infected tuberculous empyema is a surgical emergency. Unlike other surgical emergencies (abdominal) the urgency is one not of hours but of days. Surgical intervention should be instituted as soon as possible after the diagnosis has been made. Thoracoplasty should be started before the patient loses his resistance, even in the presence of fever and toxicity, repeated aspirations being employed to control toxicity. It is our belief that thoracotomy as a preliminary operation should be performed only when the empyema is fetid, containing anaerobic organisms. Thoracotomy should be done only after the pulmonary cavity has been collapsed by one or two stages of thoracoplasty. We delay thoracotomy so as to prevent spread from a re-opened cavity, as we have mentioned above (Fig. 4). In those cases in which a sinus persists after thoracotomy, phrenic exersis may be performed as it not infrequently aids in the closure of the remaining empyema space.

SUMMARY

1. Thirty-seven cases of tuberculous empyema, pure and mixed, are analyzed and classified.

2. Eighty-one per cent of the empyemas studied were secondary to induced pneumothorax.

3. Mortality was 72.3 per cent.

4. A comprehensive classification of tuberculous empyema is offered.

5. The urgency of early surgical intervention in the mixed type of tuberculous empyema is pointed out.

6. The danger of prolonged conservative treatment in the pure types of tuberculous empyema is demonstrated.

7. The advisability of performing early thoracotomy in the presence of the mixed type of infection is questioned.

8. Early thoracoplasty seems advisable.

9. A review of the literature is presented.

Seven of our cases were studied at the Mount Sinai Sanatorium, Prefontaine, Province of Quebec. Those cases of surgery reported were operated upon in Dr. Fraser Gurd's service at the Grace Dart Home Hospital.

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INFECTIONS OF MENINGES AND BRAIN OF PHARYNGEAL ORIGIN

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INFECTION of the meninges and brain originating from the pharynx occurs in 2 ways: (1) directly, by extension of a phlegmon of the parapharyngeal space, and (2) indirectly, by way of the blood stream, as in general sepsis. Each mode will be discussed separately and illustrative cases presented.

INFECTION OF THE MENINGES BY MEANS OF A PHLEGMON OF THE PARAPHARYNGEAL SPACE

This avenue of infection is well illustrated in a case which is of particular interest not only because of its uncommon occurrence, but especially because it was possible to reconstruct a puzzling sequence of clinical events by an extensive gross and microscopical study of postmortem material. That the clinical picture at the beginning was so obscure as to prevent the institution of timely surgical measures merely reflects again the guile with which disease processes betray the clinician.

CASE 1. A female, age 49 years, experienced intense pain in the right side of the neck on swallowing. The pain was first noticed when she awoke on December 13, 1937. There was marked tenderness over the right side of the neck. As the pain became progressively worse a laryngologist was consulted in the afternoon of the same day. He found the right palatopharyngeal arch to be markedly edematous, and the lingual surface of the epiglottis on the right side and the right aryepiglottic fold similarly affected. The edema appeared to be non-inflammatory in character and suggestive of an angioneurotic edema (Quincke). Palpation elicited tenderness along the right side of the neck at the level of the thyroid cartilage. A tentative diagnosis of angioneurotic edema was made, inasmuch as the patient had such a condition 4 years previously. At that time the condition disappeared within 1 day. Predicating against the foregoing diagnosis at this time, however, was a temperature of 102 degrees F. Ten cubic centimeters of calcium was injected intramuscularly and an adrenalin spray was prescribed. There was only slight improvement and the patient was subsequently admitted to the ear, nose and throat department of the Polyclinic in Vienna on December 14, 1937.

The patient experienced one chill on the day of her admission, but the tenderness of the neck and edema of the right palatopharyngeal arch diminished. At the same time, however, the edema of the entire epiglottis was found to be increased and appeared to be more of an infiltrative character. Laboratory findings: white blood cells, 14,500; polymorphonuclears, 85 per cent; immature cells, 5 per cent; lymphocytes, 4 per cent; monocytes, 5 per cent; eosinophiles, 1 per cent. Urine: albumen, 25; urobilinogen, increased. Microscopic examination revealed: bacteria, leucocytes, and erythrocytes. Roentgenogram (Fig. 1) showed extensive swelling of the upper portion of the epiglottis, aryepiglottic folds, and around the arytenoid cartilages. The valleculæ were distended from below; in their ventral regions a number of rarified spots were seen, the borders of which were irregular and extended downward to the level of the anterior surface of the body of the hyoid bone. This was thought to be either an ulcer or an abscess.

Because of the massive swelling, the valleculæ were hidden from view by the apposition of the base of the tongue to the lingual portion of the epiglottis. The free portion of the tongue moved readily and was not swollen.

On December 15 the left submaxillary region was found to be slightly tender; the entire soft palate and uvula were swollen and wax-like in appearance; edema of palatopharyngeal arch was gone; tonsils were normal in appearance; and the epiglottis was extensively infiltrated so that it was "omega shaped." The vocal cords were hidden from view by the epiglottis. The right pharyngo-epiglottic fold pre-

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Fig. 1. Extensive swelling of the upper portion of the epiglottis, aryepiglottic folds, and around arytenoid cartilages. *F*, Floor of mouth; *a*, clear spots; *E*, epiglottis; *Z*, hyoid bone; *A*, arytenoid cartilage; *C*, thyroid cartilage.

sented a yellow area, probably a submucous abscess; the left pharyngo-epiglottic fold was thickened. The base of the tongue was greatly swollen; there were no chills. A treatment with a heat lamp was given.

On December 16, pain on the opposite side (left) appeared for the first time and tenderness of the left submaxillary region was found to be present. The epiglottis remained the same as on the previous day. The base of the tongue was swollen, more on the left than on the right side. Edema of the palatine arches and soft palate had disappeared by this time. On the left side of the uvula and on the left lateral wall of the pharynx a defect of the epithelium was seen.

On December 17, trismus of moderate degree was present. The inferior pole of the left parotid gland was swollen. The epiglottis was firmly infiltrated. Tenderness of the left side of the neck in the supraclavicular region, region of the jugular veins, and the region of thyroid cartilage was elicited. Because of the diffuse tenderness and relatively slight rise in temperature to 100 degrees F., operation was postponed until the next day. On December 18, the patient was pale and slightly cachectic. A cardiac rate of 88 with a compressible pulse was present. The posterior part of the larynx was visible. At this time the swelling of the epiglottis and the left lateral wall of the pharynx was of moderate degree; whereas the swelling in the region of the left parotid gland was increased, but no fluctuation was present. During the afternoon of the previous day the whole left side of the neck and the superior part of the thorax were tender and slightly infiltrated. At this time the region of the jugular vein was not at all tender, but the supraclavicular fossa was slightly painful to palpation.

The following indicated the need for operation: (1) cachectic appearance, (2) compressible pulse, and (3) increased swelling in the region of the parotid gland. Unfortunately, operation was postponed because of the subnormal temperature of 97 degrees F. and because the consulting internist found the pulse good and considered it to be a case of parotitis epidemica. In fact, some of the people living at the house of the patient had such a condition at the same time. On December 19, the parotid area was more swollen. A solid infiltration extended downward to the level of the thyroid cartilage and continued from there along the sternocleidomastoid muscle up to its insertion at the mastoid process. A circumscribed infiltration in the region of the left sternoclavicular joint was present. The infiltrated area was tender but not fluctuant. The larynx was not changed. Trismus was moderate and the patient showed a definite pallor.

The temperature was 96.8 degrees F. for the entire day. On December 20, the scleræ were slightly yellow; the lips were dry and covered with crusts. The temperature was 98 degrees F. The left parotid was greatly swollen. The swelling extended into the posterior part of the neck to the clavicle and forward to the anterior border of the masseter muscle. The swelling throughout had sharp borders, except for the region of the clavicle. The auricle was in normal position. No paresis of the facial nerve was present and there was no fluctuation. Blood count showed 25,800 leucocytes; polymorphonuclears 79 per cent; juvenile forms, 16 per cent; lymphocytes, 1 per cent; monocytes, 4 per cent; toxic granulations and many thrombocytes. The Wassermann reaction was negative.

At operation (H. Brunner) an incision was made along the sternocleidomastoid muscle from the mastoid process to the level of the cricoid cartilage. An abscess was found in the fascia colli in front and within the muscle. Exploration on the anterior border of the muscle revealed a cavity filled with thin, fetid pus. Tissue in the surrounding areas was necrotic. The parapharyngeal space along the blood vessels could be entered with the gloved finger. Pus flowed freely. The sheaths of the vessels were slightly thickened but otherwise normal. The jugular vein, which was normal, was exposed for about 1.5 centimeters for the purpose of ligation at a later date if necessary. Jugular lymph nodes were swollen and there was some evidence of a black discoloration. As a final step the anterior mediastinum was exposed and revealed an edematous, necrotic tissue. No pus was found. Loose tamponade of the anterior mediastinum was carried out. An iodoform gauze drain was introduced into the parapharyngeal space.

The pus contained erythrocytes and a scanty mixture of bacteria with gram-positive diplococci which

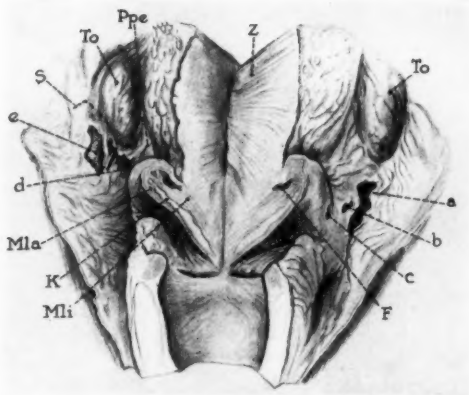

Fig. 2. Condition present after opening the larynx. *Z*, tongue split in the midline; *To*, tonsils; *a*, *b*, and *c*, ulcers in the lateral wall of the pharynx on the right side; *F*, fistula piercing the mucous membrane on the lingual side of the epiglottis, *Mli*; *K*, cartilage of epiglottis; *Mla*, mucous membrane on the laryngeal surface of epiglottis; *d* and *e*, ulcers in the lateral wall of the pharynx on the left side; *S*, probe passing the fistula from the lateral wall of the pharynx into the parapharyngeal space; *Ppe*, plica pharyngo-epiglottica.

Fig. 3. System of channels produced by phlegmon. *a*, Probe passing from the abscess within the parapharyngeal space to the base of tongue; *Z*, hyoid bone; *S*, thyroid cartilage; *F*, fistula piercing the lingual mucous membrane of epiglottis filled with barium.

appeared like pneumococci. Fine gram-negative bacilli prevailed. The culture revealed Bacterium coli commune, which at first grew anaerobically and later, after more implantations, aerobically. After operation on December 21 a slight general jaundice appeared. The wound was dry and showed diffuse necrosis but no pus. A slight euphoric state was noted. The swelling of the parotid gland, tenderness, and edema over the clavicle were not changed. Blood count showed 26,000 leucocytes; polymorphonuclears, 78 per cent; juvenile forms, 14 per cent; lymphocytes, 2 per cent; monocytes, 6 per cent; toxic granulations and toxic forms of nuclei. A blood transfusion was given. From time to time the patient was delirious. On December 22, progressive weakness was noted. In the evening she became unconscious and died.

Autopsy was performed on December 23. The dura was well stretched; the leptomeninges were delicate and very hyperemic. A hyperemia of high degree and petechial bleeding was present in the leptomeninges over the pons.

No abnormal contents were present in the abdomen. Both lungs evidenced slight adhesions onto the diaphragm, but otherwise were normal. The heart was the size of the fist of the cadaver. The tip of the heart was formed by the left ventricle. The wall of the left ventricle was approximately 12 millimeters thick and the wall of the right ventricle 4 millimeters thick. The heart muscle was very flaccid and had the color of loam. All parts of the heart were dilated. The valves, coronary arteries, and the great arteries of the body were normal. The liver had a normal form; its parenchyma was lacerable and appeared cooked. The spleen was enlarged, soft, and its mass soft. The cortex of the suprarenal glands had no lipoid deposit. Red patches were found in ascending groups in the right kidney. Red stripes extended from these patches and converged to the pyramids.

The following organs were given to the author for investigation: (1) The organs of the neck. (2) A frontal disc, 5 centimeters thick through the base of the skull which included the ascending part of the mandible. This disc was cut anteriorly, in front of the sphenoid sinus through the posterior parts of the nose, the soft palate, and the junction of the horizontal and ascending parts of the mandible, and posteriorly, through the most anterior part of the petrous bones and the apex of the pyramid, downward along the anterior surface of the spine.

Diagnosis. Multiple abscesses of the base of the tongue followed by a phlegmon of the lateral triangle of the neck which extended into the parotid region and the pterygopalatine fossa; internal, suppurative, acute pachymeningitis in the region of the operculum sellæ and clivus; parenchymatous degeneration of the viscera; right ascending pyelonephritis.

Microscopic findings of special autopsy specimens. The following were normal: left submaxillary and lingual glands, floor of the mouth, large vessels of the neck, and the surrounding area on the right side.

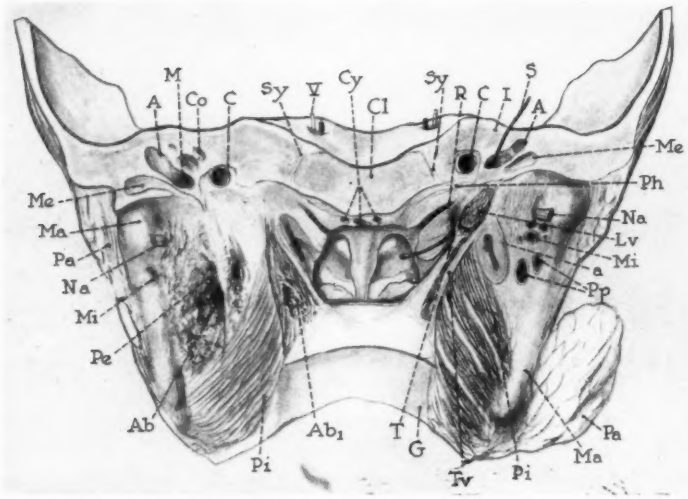

Fig. 4. Diagram of the posterior surface of specimen. *A*, antrum mastoideum; *M*, middle ear; *Co*, cochlea; *C*, internal carotid artery; *Sy*, synchondrosis petro-occipitalis; *V*, trigeminal nerve; *Cy*, small cysts within the mucous membrane on the roof of the pharynx; *Cl*, clivus; *I*, bony capsule of the cochlea; *S*, probe passing through the tube and the middle ear; *Me*, auditory external meatus; *Ph*, cut through the mucous membrane on the posterior wall of the pharynx; *R*, groove of Rosenmueller; *Na*, nervus alveolaris; *Lv*, levator veli palatini muscle; *Mi*, arteria maxillaris interna; *a*, circumscribed inflammation within the parapharyngeal space; *Pp*, veins of the pterygoid plexus; *Tv*, tensor veli palatini muscle; *T*, eustachian tube; *Pi*, pterygoideus internus muscle; *Ma*, mandible; *Pa*, parotid gland; *G*, soft palate (cut through); *Ab*, *Ab*$_1$, abscesses within the parapharyngeal space; *Pe*, pterygoideus externus muscle.

The left side of the neck revealed a recent, unencapsulated abscess about the size of a bean along the posterior wall of the internal carotid artery between the jugular vein and carotid artery corresponding to the position of the sinus caroticus. The abscess reached the vagus but did not invade it. The lumen of the great vessels in the tissue surrounding the abscess was normal. The location of this abscess corresponded to the place where the sheath of the vessels had been opened at the time of operation.

The larynx was opened by a median incision reaching toward the hyoid bone and through the middle of the base of the tongue. After the larynx was opened the condition as shown in Figure 2 was observed. The sectioned base of the tongue, *Z*, and both tonsils, *To*, were seen. Behind the right tonsil in the lateral wall of the pharynx (lateral part of the pharyngo-epiglottic fold) there was an ulcer, *a*, about 1 centimeter long and 4 millimeters wide. The ulcer had sharp, irregular, and undermined margins and a base composed of necrotic and purulent tissue. This ulcer was sharply demarcated from its surroundings and had no communication with the parapharyngeal space. Mesial to this ulcer was a much smaller one, *b*, similar in appearance to the first. By means of a fistula covered with mucous membrane this ulcer led into a third ulcer, *c*. The latter was just a pinhead opening in the mucous membrane of the most superior part of the aryepiglottic fold. A probe introduced into this opening entered a larger cavity, which communicated with a fistula, *F*, which penetrated the epiglottis. This fistula was found in the greatly swollen, hemorrhagic, lingual, mucous membrane of the epiglottis, *Mli*. The cartilage, *K*, and the lingual, mucous membrane of the epiglottis, *Mla*, were normal. The fistula, *F*, emptied into a large ulcer, *d*, situated in the left pharyngo-epiglottic fold, *Ppe*. This ulcer, *d*, was separated from another ulcer by a narrow bridge of mucous membrane. These ulcers, *d*, *e*, were also of the same appearance as the first ulcers, *a*, *b*, and *c*. When a probe, *S*, was introduced into the ulcer, *d*, it penetrated without resistance into the parapharyngeal space. In that space a recent abscess was present, located above the lingual artery and stylopharyngeal ligament, and between the wall of the pharynx and the stylopharyngeal muscle.

In order to get a clear view of the system of channels produced by the phlegmon at the base of the tongue, the entire fistula was filled through the ulcer, *c*, with barium paste. A roentgenogram of this specimen was then taken (Fig. 3). This picture

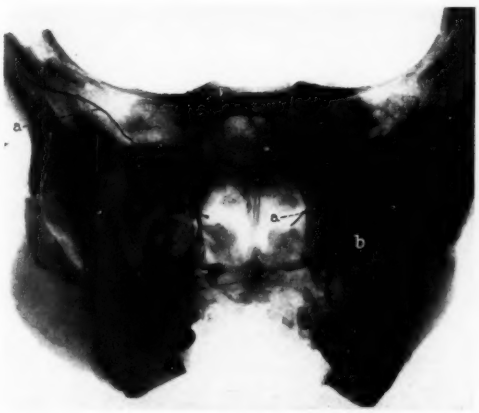

Fig. 5. Large abscess in the parapharyngeal space between the ascending branch of the mandible and the pterygoid process. *a*, Probes in the eustachian tubes; *b*, abscess within the parapharyngeal space filled with barium.

Fig. 6. Extension of the abscess sagittally. *a*, Probes in the eustachian tubes; *A*, abscess filled with barium.

shows the fistula extending above the hyoid bone around the epiglottis as a band about ½ centimeter long and rather sharply bordered. Only in the midline is the barium a little irregularly outlined over the cut surface, so that the margins are somewhat effaced. The probe is seen to lead from the ulcer, *d*, (Fig. 2) into the parapharyngeal space. This represents the route of the phlegmon of the tongue to the parapharyngeal space.

The phlegmon did not extend caudalward from the place of perforation. On the contrary, the phlegmon continued within the parapharyngeal space upward as is shown clearly in the second specimen. The anterior borders of the specimen are through the posterior ethmoid cells, the posterior part of nose, and the posterior wall of both maxillary sinuses. This entire area was of no significance. Of great significance is the posterior surface of the specimen (Fig. 4). The specimen was prepared as follows: After it was learned that the retropharyngeal space was normal, the posterior wall of the pharynx, *Ph*, was cut off at its insertion at the roof of the pharynx. The soft palate, *G*, had already been cut off at autopsy. The roof of the pharynx presented remains of lymphatic tissue and some mucin filled cysts, *Cy*. The muscles of the palate on the right side were dissected, the levator veli palatine, *Lv*, was cut, and the tensor veli palatini, *Tv*, was left untouched. Finally, the posterior walls of the tube, *T*, were cut away on both sides so that both tubes could be probed. The probe on the right side entered the middle ear but this was not possible on the left side. The mucous membrane of the cartilaginous tubes was found to be normal. By further dissection, the following was found: The marrow of the clivus, *Cl*, was normal. The dura over the clivus was covered by some thick pus; on each side the inferior petrosal sinus was located. Farther forward the branches of the trigeminal, *V*, and its accompanying veins perforated the dura. The petro-occipital synchondrosis, *Sy*, connected the clivus with the petrous bones. Here both internal carotid arteries, *C*, were seen. On the right side the most anterior part of the cochlear capsule, *I*, and on the left, the most anterior part of the bony cochlea, *Co*, the middle ear, *M*, the most anterior part of the attic, *A*, and the anterior wall of the external auditory meatus, *Me*, were located. Below the base of the skull were seen the ascending ramus of the mandible, *Ma*, and more lateralward, the remains of the parotid glands, *Pa*. The cut wall of the pharynx, *Ph*, and the Rosenmueller fossæ, *R*, the cartilaginous portions of the eustachian tubes, *T*, are seen in the middle. The area, bordered mesialward by the processus pterygoideus and lateralward by the mandible, was of particular interest. On the right side the bundles of the internal pterygoid muscle, *Pi*, and imbedded in fat and loose connective tissue, the auriculotemporal nerve, *Na*, the internal maxillary artery, *Mi*, with some small accompanying veins and some larger but not thrombosed veins, *Pp*, belonging to the pterygoid plexus were located. At *a* there was a small vein surrounded by a peculiarly condensed tissue. This was examined microscopically.

Very marked changes were present in the left parapharyngeal space. The auriculotemporal nerve, *Na*, and the internal maxillary artery, *Mi*, are on this side, too, but the accompanying veins of the artery and the big veins of the pterygoid plexus on this side were involved in an abscess, *Ab*, about the size of an olive, situated between the internal pterygoid muscle, *Pi*, and the pterygoid process. This abscess perforated the internal pterygoid muscle,

Fig. 7. Right side. *A*, anterior; *p*, middle fossa; *D*, dura; *a*, thrombophlebitis purulenta of veins within the dura; *F*, fracture in the posterior frame of the foramen ovale; *AM*, abscess within a marrow space; *Fo*, foramen ovale; *W*, trigeminal nerve; *Sym*, sympathetic nerve; *Te*, tensor veli palatini muscle; *T*, cartilaginous tube; *L*, levator veli palatini muscle; *Pc*, thrombophlebitis purulenta of veins of carotid plexus; *C*, internal carotid.

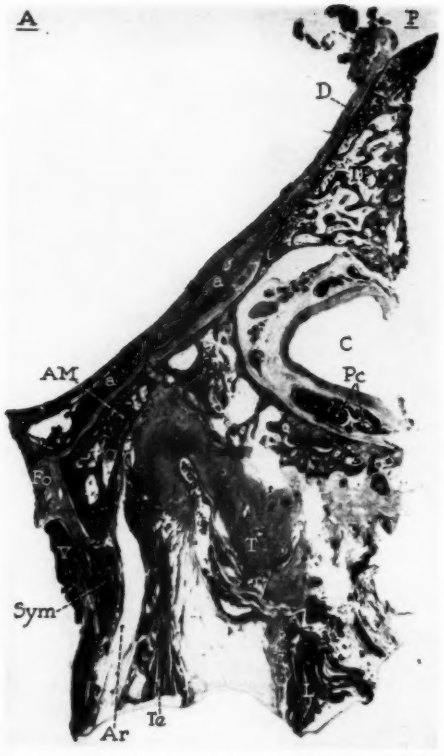

Fig. 8. Right side, *A*, Anterior; *p*, middle fossa; *D*, dura; *TPy*, tip of the pyramid; *a*, thrombophlebitis purulenta of veins within the dura; *AM*, abscess within the marrow space; *Fo*, foramen ovale; *V*, trigeminal nerve; *Sym*, sympathetic nerve; *Ar*, artefact; *Te*, tensor veli palatini muscle; *T*, cartilaginous tube; *L*, levator veli palatini muscle; *Pc*, thrombophlebitis purulenta of veins of plexus caroticus; *C*, internal carotid artery.

Ab, as well as the levator veli palatini muscle in a mesial direction against the wall of the pharynx. The left maxillary artery and the inferior part of the external pterygoid muscle were examined microscopically.

In order to observe the extension of the abscess the cavity was filled with cotton pads soaked in barium paste. Figure 5 shows a large abscess in the parapharyngeal space between the ascending branch of the mandible and the pterygoid process. This abscess did not reach the base of the skull although the cotton pad was packed into the cavity with pressure. Figure 6 shows the extension of the abscess sagittally. The abscess did not reach the pterygoid process but approached the bony tube. Finally, both pictures show that the marrow of the basilar bone was normal, that the right tube was patent in its extent, and that the left tube was obstructed at the junction of the cartilaginous, with the bony, tube.

After sawing through the skull in the midline, we found the sphenoid sinus and the marrow of the sphenoid body to be normal. The left cavernous sinus contained much pus; there was no frank pus found within the right sinus. The left cavernous sinus contained the same bacteria as those found in the parapharyngeal abscess, i.e., bacteria similar in appearance to those of the flora of the mouth, mixed with streptococci and a great number of fusiform bacilli.

Microscopic examination. *1. The parapharyngeal space on the right side* (Fig. 5, *a*). A diffusely infiltrated fatty tissue containing small veins was seen. The walls of these veins were infiltrated with purulent material. This infiltrate approached the endothelium and destroyed it in places; at other points, the elastic layer as well as the endothelium showed evidence of involvement.

Thrombi and collections of pus were seen within the veins. Purulent, strip-like infiltrations starting from the veins invaded the surrounding fat. The small arteries were surrounded by pus but their walls were normal. Finally, the sheaths of the nerves were infiltrated but the nerves themselves were found to be normal.

Fig. 9. Right side. *A*, Anterior; *p*, middle fossa; *D*, dura; *a*, thrombophlebitis purulenta of veins within the dura; *T*, cartilaginous tube; *Am*, middle meningeal artery within the foramen spinosum; *Tp*, thrombophlebitis purulenta of veins of plexus pterygoideus; *AM*, abscess within a marrow space; *Te*, tensor veli palatini muscle; *L*, levator veli palatini muscle; *Pc*, thrombophlebitis purulenta of veins of carotid plexus; *C*, internal carotid artery; *Ca*, calcification of media of internal carotid; *Co*, bony capsule of cochlea.

Fig. 10. Right side. *D*, Dura; *Pp*, thrombophlebitis of plexus pterygoideus; *a*, posterior border of foramen spinosum; *c*, anterior border of foramen spinosum; *b*, anterior and inferior border of foramen spinosum; *Am*, middle meningeal artery within the foramen spinosum.

2. The apex of the right temporal bone. The intima of the internal carotid artery was slightly thickened; the elastic layer was dissected and in some places the media was calcified (Fig. 9). In the tissue surrounding the carotid artery, the carotid plexus was extensively thrombosed and abscessed (Figs. 7 and 8). This purulent thrombophlebitis decreased in extent as the plexus was traced from the cavernous sinus to the knee of the carotid artery (Fig. 11).

The cartilaginous and bony tubes and their lumina were normal except for some hyaline degeneration in the mucous membrane of the bony tube.

The apex of the pyramid (Fig. 8) was diploetic and contained loose spongy bone, the marrow of which was partly lymphatic and partly fatty. For the most part, the marrow was normal.

The dura of the middle fossa showed an extensive, intradural and extradural suppurative process (Figs. 7, 8, 9 and 11). The extradural suppuration, in many places, had broken into the marrow spaces of the tip of the pyramid and there produced abscesses of various sizes. Some bundles of the trigeminal, that are partly within and partly beyond the dura, were surrounded or destroyed by the suppuration. As in the case of the thrombophlebitis of the carotid plexus, intradural suppuration decreased lateralward in extent. In the lateral part of the middle fossa were many Pacchionian granulations and brain hernias, neither showing inflammatory changes.

The levator veli palatini muscle, fat tissue, and dense connective tissue (Figs. 8, 9, and 11) were seen posterior to the cartilaginous tube where it is fixed to the inferior surface of the petrosal bone. Within that fat and connective tissue are arteries of middle size which were normal, and many glands and excretory ducts of glands which probably belong to the glands of the pharyngeal mucous membrane. There were dense piles of leucocytes (periglandular abscesses) around some of these glands.

Anterior to the tube are the tensor veli palatini muscle and that part of the parapharyngeal space which lies just below the base of the skull (Figs. 9 and 10). In that space lie the mandibular branch of the trigeminal before it enters the foramen ovale, and the middle meningeal artery as it enters the foramen spinosum. There was an extensive intradural suppurative process where the dura covered the bony margins of the foramen ovale (Figs. 7 and 8*a*). The intradural infection extended farther by breaking through the posterior wall of the foramen ovale into the spongy bone and produced a big abscess within the marrow (Figs. 7 and 8 *Am*). At another site a large fistula was seen in the posterior wall of the foramen ovale (Fig. 7 *F*). Both margins of this fistula were dislocated and showed evidence of Howship's lacunæ but no osteoclasis. The opening of the fistula was filled with scar tissue consisting of many cells. At this place, although the suppuration had broken through the posterior wall of the foramen ovale in the direction of the cartilaginous tube, this particular fistula was already healed at the time of the patient's death. In spite of the extensive suppuration in the region of the foramen ovale, no evidence of suppuration was seen within the branches of the trigeminal that pass through the foramen.

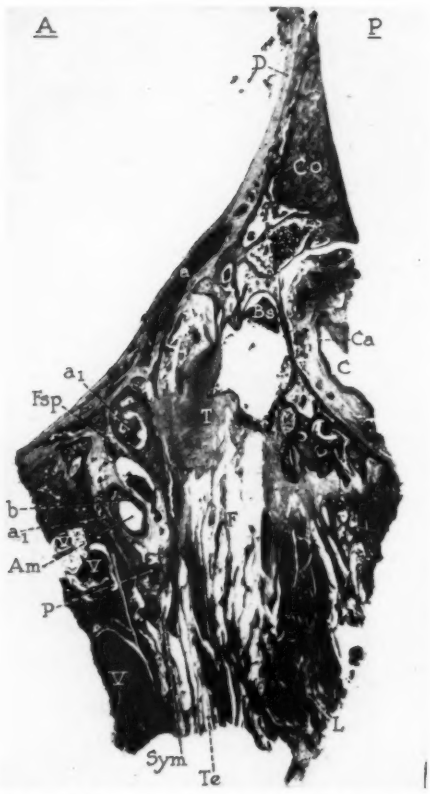

Fig. 11. Right side. *D*, Dura; *Co*, bony capsule of cochlea; *a*, thrombophlebitis purulenta of veins within the dura; *a1*, thrombophlebitis of veins of pterygoid plexus; *Fsp*, foramen spinosum; *b*, purulent thrombus within the middle meningeal artery, *Am*; *V*, trigeminal nerve; *P*, pus within the parapharyngeal space; *Sym*, sympathetic nerve; *Te*, tensor veli palatini muscle; *L*, levator veli palatini muscle; *F*, fat within the parapharyngeal space; *T*, cartilaginous tube; *C*, knee of internal carotid artery; *Ca*, calcification of tunica media of internal carotid artery; *Bs*, bony splits within the tube.

A different sort of process was observed in the neighborhood of the foramen spinosum (Figs. 9, 10, and 11). The middle meningeal artery was normal, and the branches of the sympathetic nerve, which accompany the artery, were almost normal. But in the loose connective tissue surrounding the artery there was an extensive abscess, the result of a purulent thrombophlebitis. This abscess undoubtedly was a continuation of an abscess within the parapharyngeal space which extended through the foramen spinosum into the cavity of the skull. Therefore, the infection of the dura on the right side can be said to have originated therefrom.

3. The ulcers in the right lateral wall of the pharynx (Fig. 2). In some places these ulcers reached the muscle layer of the pharynx. There was an inflammatory infiltration of moderate intensity in the connective tissue septa between the muscle bundles of the superior pharyngeal constrictor.[1]

4. Parapharyngeal space on the left side. There was a suppurative thrombophlebitis of the small veins. The fat and the loose connective tissue on that side was more affected than on the right side. In addition to the ribbon-like infiltrations, considerable necrosis and masses of bacteria, hemorrhages, and abscesses were noted. The purulent infiltration reached the media of the internal maxillary artery. The connective tissue sheaths of the nerves were infiltrated, but the nerves themselves were almost all normal.

5. Region of the left pterygoid process. The veins of the pterygoid plexus were seen within the fat and loose connective tissue that surround both pterygoid processes. These veins contained either purulent thrombi or were thoroughly destroyed to form small and large abscesses. The large veins of the pterygoid plexus were, on the whole, normal. The terminal branches of the internal maxillary artery were also normal. It is surprising that the perineural tissues showed no, or very little, inflammatory change. The bundles of the pterygoid muscles were for the most part normal.

The marrow of the pterygoid process was partly fatty and partly mixed; small abscesses were seen here and there. The bone and the pterygomandibular ligament were more resistant to suppuration, so that the ligament was not perforated, and the bone was perforated only in a few places.

6. The apex of the left temporal bone. The changes here were essentially the same as on the right side, except that they were more extensive (Fig. 12). The internal carotid artery showed a thickening of the intima and a calcification of the media. The thrombophlebitis around the artery was so extensive that in many places there were no veins at all but only frank pus. In many places the pus invaded the artery (Figs. 12 and 13*a*). At these places the nuclei in the wall of the artery did not take the stain; elsewhere the endothelium was destroyed. The lumen of the artery contained a mural thrombus (Fig. 12*Th*). The extensive peri-arterial suppuration continued also along the knee of the internal carotid artery into that part of the artery that descends into the neck.

The bony tube was normal and also the cartilaginous part with the exception of the region of the isthmus of the tube. Under normal circumstances the mucous membrane is adjacent to the cartilage; villi of the mucous membrane are seldom seen. In

[1] A study of fistulous tracts was made by Laskiewicz who filled emptied cavities following peritonsillar abscesses with radiopaque fluids and subsequently examined these roentgenologically. He never could find any perforation of the lateral wall of the pharynx in these cases. The lateral wall of pharynx was only pushed lateralward. His findings prove very little, since infection can also reach the parapharyngeal space through the pharyngeal constrictor, without leaving behind any patent fistula.

this case, the mucous membrane was in many folds which greatly narrowed the lumen. On some places that narrow opening was entirely closed by a serous exudate which developed in the following way: Under normal circumstances some cones containing connective tissue and blood vessels invade the cartilage of the tube, perforate the cartilage, and communicate with the subepithelial tissue of the mucous membrane. In our case, the blood vessels in these cones were hyperemic, at times also infiltrated, which gave evidence of their rôle in the transportation of infection, without affecting the cartilage, from the surrounding tissue into the mucous membrane of the tube. The marrow in the tip of the petrous bone was mixed. In many marrow spaces the marrow consisted of only lymphoid tissue which was a consequence of inflammation.

The dural veins were extensively thrombophlebitic and purulent so that big intradural abscesses were seen in a number of places. Extradurally, there was not much pus present. The superior petrosal sinus was thrombosed. There were many Pacchionian granulations in the dura of the lateral part of the middle fossa, but these were neither inflamed nor infiltrated. The branches of the trigeminal nerve, which perforate the dura, were also slightly inflamed. Only little pus was to be found in the Meckel's cavity. The gasserian ganglion was practically normal.

In that part of the parapharyngeal space, which is situated before the tube and before the tensor veli palatini muscle, big abscesses were seen and an extensive thrombophlebitis of the pterygoid plexus. These abscesses also surrounded the branches of the trigeminal and the sympathetic nerves; generally they did not invade these nerves. There was a split-like abscess in one place coming from the parapharyngeal space and traversing the trigeminal and adjacent sympathetic ganglion. The suppuration in the parapharyngeal space reached the ala magna of the sphenoid bone on the one hand, and on the other invaded the posterior wall of the foramen ovale and produced big abscesses in the marrow of the bone.

In the opening of the foramen ovale there was only a slight inflammatory change within the loose connective tissue between the bundles of the third branch of the trigeminal. However, there was an extensive, purulent periphlebitis which accompanied the meningea media artery through the foramen spinosum. Thromboses seldom occurred. In the opening of the artery mural piles of leucocytes were found which lay in a network of fibrin. At these places the endothelium layer was almost absent which indicated the beginning of thrombosis. Otherwise, the wall of the artery and the bony walls of the foramen spinosum were normal.

The mucous membrane in the anterior part of the middle ear was hyperemic and inflamed. The well developed, pneumatic cells in the solid angle of the pyramid was lined principally with normal mucous membrane and only partly with edematous mucous membrane.

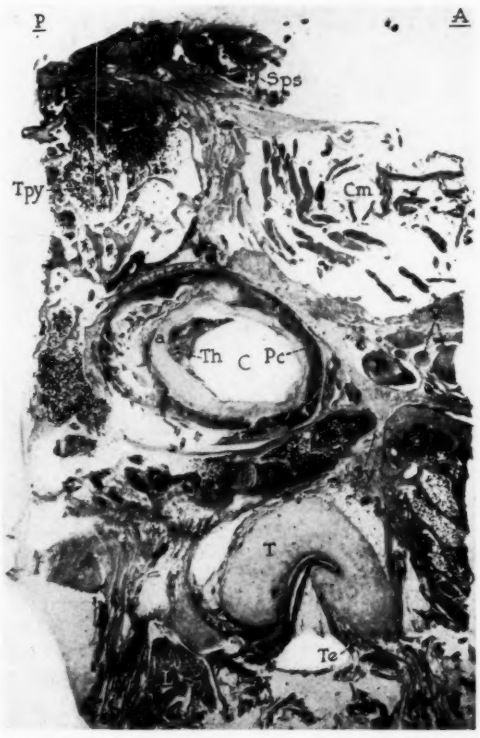

Fig. 12. Left side. *Sps*, Sinus petrosus superior (thrombosed); *Cm*, cavum meckeli; *Pc*, thrombophlebitis purulenta of veins of carotid plexus; *V*, trigeminal nerve; *P*, abscess within the dura; *Sy*, syndesmosis sphenopetrosa; *Te*, tensor veli palatini muscle; *T*, cartilaginous tube; *L*, levator veli palatini muscle; *Th*, thrombus within the internal carotid artery, *C*; *Tpy*, tip of the pyramid; *a*, space shown in Figure 13 in higher power; *A*, anterior; *P*, middle fossa.

7. Cavernous sinus on the left side. There was an extensive suppurative process reaching the media of the internal carotid artery. The media itself was normal. The connective tissue sheath of the nerves was only slightly infiltrated, and some small branches of the nerves were invaded by leucocytes.

It is obvious that in cases like these the most exact macroscopical examination is not sufficient to visualize the extent of the pathological processes. The microscopical examination of tissues which seem macroscopically normal is absolutely necessary. Consequently, quite enormous changes were discovered, the course of which can be traced only when cor-

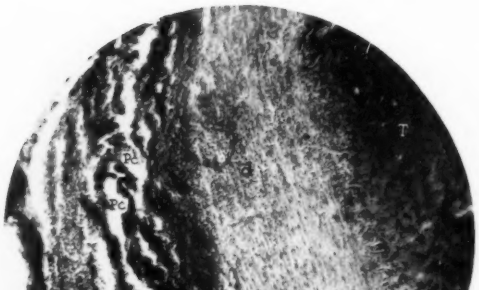

Fig. 13. The space, *a*, in Figure 12 in higher power. *a*, *b*, internal carotid artery; *d*, wall of the artery infiltrated; *c*, margin between the infiltrated wall (upward) and the necrotic wall (downward); *Pc*, thrombophlebitis purulenta of the carotid plexus; *T*, purulent thrombus.

related with the clinical features of the case. The patient's illness began with a phlegmon in the right half of the base of the tongue and in the neighboring lateral wall of the pharynx. Later, the phlegmon extended along the base of the tongue and wandered from the right to the left. This explains the presence of ulcers in the lateral wall of the pharynx on both sides. These ulcers were connected by a fistula piercing the base of the tongue. The ulcers on the right side did not show a perforation of the constrictor pharyngis, but on the left side an invasion of the parapharyngeal space undoubtedly took place. Consequently, large abscesses developed in the loose connective tissue of the left parapharyngeal space, as shown in Figure 4. From these abscesses, the infection continued partly downward and partly upward. The continuation of the infection downward followed the sheath of the vessels and might have been stopped by the operation. Of greater importance was the continuation of the infection upward into the cavity of the skull. Frank and Scheer studied the pathways of such infections and found it possible for the extension to proceed (1) along the veins in the form of a purulent thrombophlebitis (as a rule the order is as follows: veins of the pterygoid plexus, to the veins of the foramen ovale, and then to the cavernous sinus); (2) directly, as a phlegmon along the sheaths of the vessels and nerves into the foramina ovale, lacerum, and spinosum; and (3) by an osteomyelitic erosion of the bony base of the skull.

In this case there was purulent thrombophlebitis of the pterygoid plexus and of the veins in the foramen ovale, a slight infiltration of the sheaths of the mandibular branch of the trigeminus, an osteomyelitis of the posterior wall of the foramen ovale, and, finally, an extensive thrombophlebitis of the venous plexus around the carotid artery; in one word, all pathways, mentioned by Frank and Scheer were affected. It is important that the walls of the eustachian tube, which were surrounded everywhere by pus, were not affected. The tube reacted only with the production of a little serous exudate filling up the lumen of the tube in the region of the isthmus and with the swelling of its mucous membrane which obstructed the passage of a probe on the left side. The wall of the carotid artery in the carotid canal was infected and the lumen at that place contained a mural thrombus.

The condition was too far advanced to permit a definite decision as to which pathway the infection followed to reach the endocranium. It is the writer's belief in this respect that the thrombophlebitis of the veins of the pterygoid plexus might have been of greatest importance. When it is recalled that in the microscopic specimen pus was seen within and around the vessels, it cannot be decided whether the pus had propagated upward along those channels or had sloped downward along these same channels. This question always arises in postmortem cases that have been examined. Consequently, an unquestionable route of infection to the endocranium can seldom be uncovered. At any rate, it seems that this question is of no great practical importance, as even an exact knowledge of the pathway an infection will take into the endocranium would not make a rational regimen of treatment available.

When the infection in this case reached the endocranium, it affected the veins within the

dura and the cavernous sinus. As a result of this thrombophlebitis, many intradural abscesses of various sizes originated. These intradural abscesses perforated the dura and produced extradural abscesses which finally eroded the bone of the ala magna. It is not surprising that these intradural abscesses did not perforate into the arachnoid, since, as is well known, the endothelium of the dura is highly resistant. As a result of this investigation, one observation is foremost: both sides were similarly affected, though less on the right, despite the fact that no fistula was found on the right side, either grossly or microscopically, to account for a supposedly direct extension of the lingual phlegmon into the right parapharyngeal space.

How are the pathological changes in the right parapharyngeal space to be explained? The thrombophlebitis of the right carotid plexus is most likely a continuation of the thrombophlebitis of the left cavernous sinus. The route of infection was in the following order: left cavernous sinus, intercavernous sinus, right cavernous sinus, and right carotid plexus. An infection spreads along such channels very quickly, as reported by me many years ago. This type of infection will explain why the thrombophlebitis of the right carotid plexus, which has not been as intensive as the thrombophlebitis of the left carotid plexus, decreases in the direction from the cavernous sinus to the knee of the carotid artery. This last mentioned observation excludes the supposition that the infection of the right carotid plexus took place by way of an ascending infection from the right parapharyngeal space.

Quite a number of other conditions must be considered as far as the suppuration in the right parapharyngeal space is concerned. This suppuration was undoubtedly the result of the phlegmon in the base of the tongue, which started in the right half of the tongue. That no perforation was discovered to lead from the right half of the tongue into the right parapharyngeal space proves nothing because a similar lack of a fistulous tract is often observed in parapharyngeal space infections of tonsillar origin. Further, a preceding tonsillar infection can be resolved at the time when the phlegmon, which it produced within the parapharyngeal space, is still active and progressive.

In conclusion, the course of the disease was as follows: The phlegmon of the base of the tongue on both sides perforated into the parapharyngeal space, on the left side by an obvious fistula, but on the right by a fistula already healed supposedly at the time of death. The infection of the left parapharyngeal space continued into the endocranium. The chief routes were surely by way of the sheath of the carotid artery and the veins of the pterygoid plexus. Within the endocranium the infection produced severe intradural suppuration and a thrombophlebitis of the left cavernous and intercavernous sinuses. The infection then entered the right cavernous sinus and penetrated farther on into the right carotid plexus. From the parapharyngeal space on the right side, the infection passed along the pterygoid venous plexus, through the foramen spinosum, and into the endocranium, too, which produced essentially the same changes as on the left side.

It is proper now to reconstruct the clinical symptoms in correlation with the virulent infection. On December 13, the patient acquired, for reasons unknown, a phlegmon of the tongue which wandered from right to left and which had been, as usual, associated with high septic temperature, moderate leucocytosis, and albuminuria. The one chill that the patient had experienced and the tenderness of the left submaxillary region were direct results of the phlegmon of the tongue. On December 17, 4 days after the onset of the disease, the temperature began to fall. At the same time 3 symptoms appeared which indicated the seriousness of the disease had they been properly appreciated: (1) trismus; (2) swelling in the region of the left parotid gland; and (3) diffuse tenderness of the left half of the neck. In retrospect, we must explain these symptoms on the basis of the infection of the parapharyngeal space which occurred 4 days after the onset of the disease. Unfortunately, we did not appreciate the value of these symptoms and did not operate at once.

The subsequent course was peculiar. Instead of the septic temperature usually encountered, the temperature fell to normal by

crisis, the local inflammatory signs of the throat improved, the swelling and the tenderness of the left side of the neck decreased, which indicated an apparent improvement of the entire condition. Nevertheless, the decision was made to operate because the patient looked sick, the pulse was soft, and the swelling of the parotid had increased. In failing to give sufficient credit to these symptoms 3 precious days were lost after the onset of the supposed infection of the parapharyngeal space. But on December 20, as soon as general decay, swelling of the parotid, and leucocytosis became increased and a slight jaundice of the scleræ appeared, the operation was performed in spite of the low temperature of 98 degrees F. At operation a purulent necrosis of the connective tissue was found which reached the parapharyngeal space. There was very little pus. After operation the temperature rose slightly and the leucocytosis increased. The progress of the disease could not be stopped, however, and 2 days after operation the patient died of sepsis. The disease lasted 10 days in all; and if we suppose that the infection of parapharyngeal space began on December 17, the inflammation in that area lasted 6 days.

When the clinical course is compared with the findings at autopsy, the case reveals a number of significant findings:

1. The infection of the parapharyngeal space can manifest itself with the symptoms of a critical fall in temperature and of increasing leucocytosis. It is well known that some forms of sepsis, and indeed the most dangerous forms, can progress without giving rise to high temperature or even slightly elevated temperature. Roenau, of my department in Vienna, described a case illustrating this fact. It is not well known that an acute sepsis such as this can also originate from the parapharyngeal space. It cannot be proved that Bacillus coli, which was found to be present, was responsible for the peculiar features of this case, although it is possible that this bacterium was the contributing factor.

Orton recently described cases in which the onset of a parapharyngeal infection was associated with a fall in temperature by lysis. I have also observed such cases. It should be noted that in our case a critical fall in the temperature signalized probably the first involvement of the parapharyngeal space.

2. The autopsy findings indicate the possibility that the infection of the right parapharyngeal space took place many days before death. This supposition is supported by the fact that the fistula in the posterior wall of the right foramen ovale was filled with young connective tissue. Surprisingly this infection of the right parapharyngeal space happened without clinical signs many days before death; symptoms such as swelling of the neck, serous catarrh of the middle ear, or trigeminal neuralgia were all prominently absent. Only trismus existed which must be explained on the basis of the infection of the left rather than of the right parapharyngeal space. The conclusion to be drawn is that infection of the parapharyngeal space can exist without early, alarming, general, and local symptoms. This fact invites the suspicion that buccopharyngeal infections involve the parapharyngeal space more often than has been our belief. This conception has already been emphasized by Cobb and certain French laryngologists. Although Uffenorde does not believe this, the above tends to support the ideas represented by Cobb.

3. Symptoms of trigeminal neuralgia were absent, in spite of the fact that the parapharyngeal spaces on both sides were infected. In fact, the fibers of the trigeminal were only slightly affected; the purulent infection was found principally around the nerve and gasserian ganglion. Of course it should be borne in mind that it is much easier to establish microscopically the basis for paralysis of a nerve than for irritation. However, it is certainly true, as Wessely remarked, that trigeminal symptoms are not present necessarily in all cases of ascending infections of the parapharyngeal space. We can make an even broader observation on the basis of this case, namely, that neuralgic symptoms can be absent even when the surrounding areas of the trigeminal nerve are slightly involved.

4. Whether or not this patient had an acute serous catarrh of the middle ear is not known, because it was not investigated. But surely a blocking of the eustachian tube on the left

side existed, as evidenced by the inability to probe through the tube in the gross specimen. This blocking was the result of an inflammation of the mucous membrane in the cartilaginous tube, and this reaction in turn produced tufts and serous exudate. By what manner did the patient acquire tubal pathology? First of all, it did not result from an ascending infection along the tube from the pharynx. In order that this mechanism be considered, the tubal inflammation in such a case of bilateral infection of the parapharyngeal space as this would have to be bilateral. Instead, it was only on the left side. The idea of Wessely, namely, that the cartilaginous tube can be compressed by a parapharyngeal abscess, is not acceptable. The chief reason against this concept is the fact that the cartilaginous tube is surrounded everywhere by loose connective tissue and by fat. Also, at the point where the tube contacts the floor of the carotid canal it is separated from the bone by a thick layer of connective tissue. Consequently, if we still suppose that an abscess in the parapharyngeal space might compress the tube, it would be most essential that such an abscess be of an enormous size and continue to subject neighboring parts to great pressure. This again is not possible since the parapharyngeal space is not an entirely closed space.

The eustachian tube changes resulted from the infection passing along the connective tissue and blood vessels, which normally perforate the cartilage of the tube and extend thereby from the parapharyngeal space into the mucous membrane.

5. Our patient had an intensive intradural suppuration, a purulent thrombophlebitis of the cavernous sinus on both sides and of the carotid plexus on both sides. All these suppurations did not produce clinical symptoms. Eagleton has emphasized the lack of outstanding symptoms in certain cases of cavernous sinus thrombophlebitis. This writer made similar remarks many years ago regarding otogenous cavernous sinus thrombophlebitis. We, therefore, must disagree in this respect with Wessely's concept that: "When the infection has spread over the base of the skull and reaches through the foramina ovale and spinosum into the endocranium, the typical symptoms of thrombosis of cavernous sinus and meningitis precipitate." Our case proves conclusively that it is not always so. It is justifiable to state that when a patient dies of sepsis, originating from the parapharyngeal space, meningitis and thrombophlebitis of the cavernous sinus can be excluded with certainty only after these parts have been examined microscopically. Following up this postulate, we should definitely prove in similar cases, more often than we have in the past, the existence or non-existence of a latent meningitis and thrombophlebitis of the cavernous sinus which ordinarily are diagnosed only as *descending* infection of the neck. It will be necessary that microscopic examinations of the dura and the sinus be made even though they appear normal.

6. Twelve cases of deep abscesses of the neck occurred after buccopharyngeal infections were treated by the writer during the last few years, with 2 fatalities. When there was a chance to operate at all (a few of these patients died very rapidly), it was customary to drain the parapharyngeal space and usually to expose the jugular veins.

The present case indicates that this surgical procedure should not be followed routinely. At the time of the neck operation, the sheath of the blood vessels was found to be normal in spite of the purulent necrosis surrounding it. Nevertheless, this normal sheath was incised to expose the jugular vein. At autopsy, just at the site of exposure of the jugular vein an abscess was found. Undoubtedly, a route was opened up by incising the sheath unnecessarily at operation. Therefore, it is quite evident that at an operation for deep abscesses of the neck a normal sheath of the blood vessels should not be incised when there is purulent necrosis of the surrounding fascia of the neck.

INFECTION OF THE BRAIN BY WAY OF GENERAL SEPSIS

The second way in which infection may lead to the endocranium is illustrated by the following case:

CASE 2. Ch. Sch., a female, 43 years old, had often had angina. On March 29, 1936, fever, chills, and a swelling of the neck appeared. It had been diagnosed as "abscess of the throat." After 2 days

the conditions improved but on the third day she ran a temperature of 100.4 to 104 degrees F. Since April 1, she could neither stand nor sit up; therefore, she was admitted to the neurological clinic. On April 2, the patient began to speak slowly and incoherently. She stretched her arms to other places than she wished to stretch them. On April 3, delirium occurred.

On April 4, her temperature rose to 104 degrees F. The soft palate on the left side bulged out. The region of the left tonsil was swollen; grimaces, dysarthria, and abducens paralysis on the left occurred. The right arm was paretic and ataxic; abdominal reflexes on the right side were diminished; both legs were paretic and ataxic; abasia; reflexes and tonus on both sides were diminished. There were no signs of affection in the pyramidal tracts. On April 6, her temperature was 101.5 degrees F. The glands in the left mandibular angle were swollen, but the vessel sheath was not tender; no trismus was present. The region of the left tonsil was very red; the left tonsil was displaced by backward pressure. The palatopharyngeal arch on the left side was very much infiltrated, and a large swelling and bulging out of the lateral wall of the pharynx extended downward to the larynx. The larynx could hardly be seen, but no edema and no disturbances of the motility of larynx were present. There were 14,850 leucocytes in the blood. There was hemiparesis on the right side but the facial nerve was intact. Paresis of the lateral movement of eyes to the left and upward was present. Language could not be understood. The cerebrospinal fluid was analyzed with the following results: Pandy, ±; Nonne-Apelt, ±; Nissl, 0. Diagnosis: tonsillogenous sepsis. Operation was refused. On April 7, swelling of the lateral wall of the pharynx increased and strabismus convergens occurred. There was no rigidity of the neck but a positive Kernig was present. In the evening the patient died.

In taking out the organs of the neck at autopsy, a very well encapsulated abscess as big as a walnut, situated behind the left tonsil, was opened. The abscess cavity was filled with thick, creamy, greenish pus. In the pus as well as in the blood, the Streptococcus hæmolyticus was found. Both tonsils were enlarged and succulent. The follicles at the base of the tongue were swollen. The mucous membrane of the pharynx was very hyperemic. Trachea and esophagus were normal. The gray substance of the striated body and of the cortex cerebri were markedly hyperemic and spotted. No encephalitis was to be found within the pons. The vessels at the base of the brain were delicate.

The diagnosis was retrotonsillar abscess of the left side, septicemia, subacute tumor of the spleen, parenchymatous degeneration of the intestines, edema and hyperemia of the brain, recent hemorrhagic nephritis, and jaundice.

Microscopic examination revealed that the tonsils were hyperplastic and infiltrated by fibrous tissue. In that fibrous tissue many hyaline spots were to be seen but no cartilage. Within the constrictor pharyngis muscle much fibrous tissue was to be seen. In all sections of the brain hyperemia was found but no encephalitis. In one section a thrombus consisting of leucocytes was identified.

In Case 2 edema of the brain was produced by a pharyngogenous sepsis. We cannot maintain, of course, that the parapharyngeal space and the meninges in this case were entirely normal, since they were not examined microscopically. However, it can be stated, that anatomically as well as clinically edema of the brain was prominent.

The finding of an edematous process of the brain in a case of general sepsis is not surprising, since we know that in the course of a sepsis of any origin the brain can be affected in different ways. By toxins as well as by relaying of cocci, hyperemia, edema, swelling of the brain, hemorrhage, encephalitis, or a brain abscess can develop. As already mentioned in our case report, an edema of the brain had developed. It is very important that this edema produced clinically only local symptoms of the posterior fossa. These symptoms were as follows: (1) grimaces; (2) dysarthria; (3) abducens paralysis on the affected side; (4) ataxia; and (5) hemiparesis on the right or healthy side. These symptoms led to the erroneous diagnosis of encephalitis of the pons.

An edema of brain some times can produce the clinical features of a disease localized within the posterior fossa. But even this finding is not surprising, as we know that sometimes a non-inflammatory hydrocephalus can simulate the symptom complex of a cerebellar tumor. Nevertheless, it is important to know that a septic edema of the brain can simulate an encephalitis or a brain abscess within the posterior fossa. The importance of this fact will be elucidated by the very different prognosis. For while a brain abscess or an encephalitis impairs the prognosis of pharyngogenous sepsis to the highest degree, this is not the case in so far as edema of the brain is concerned. We must remember that an edema of the brain can disappear if the sepsis is controlled either by surgery or by conservative treatment. The following case may prove this statement:

CASE 3. A. M., female, 22 years old, had sometimes had angina. She became sick with intensive pain in the throat on January 8, 1937. The pain was localized first on the right then on the left side of the neck. On January 15, a peritonsillar abscess was found. On January 16, the condition became worse; she felt fatigue and her vision was diminished. There was no headache, but the patient experienced vertigo when she sat up. From January 16, she had strabismus. During the first 2 days of her illness she had high fever; later her temperature was only about 98.6 degrees F. Earlier she was a little drowsy.

On January 19, 1937, temperature was normal and there was no pain in the throat. The nose was normal; there was slight peritonsillar swelling on the left side. The submaxillary glands were slightly swollen on both sides, on the left more than on the right side. The glands were not tender, and the larynx was normal. The skull was diffusely tender. All nerves were tender at their point of exit from the skull, and there was slight rigidity of the neck. Lasègue's sign was slightly positive on both sides; nystagmus of second degree to the left; nystagmus of first degree to the right; vertical nystagmus upward. Paresis of the right abducens and slight paresis of the right facial nerve (?) were noted. The tongue, when put out, deviated slightly to the left; language was slow and scanning; flexion combined. The finger-nose test showed slight ataxia. There was slight hypodiadochocinesis on the left side, but not signs of affection of the pyramidal tracts. Tendinous reflexes were slightly increased on both sides. In Romberg position the patient would fall without certain direction. The cerebrospinal fluid was clear; Pandy ±, Nonne-Apelt −, Nissl, 0.02,6/3 cells. Urine was normal. Blood count showed 9,000 leucocytes. The diagnosis was encephalitis.

On January 21, the eye grounds were normal. Abducens paresis had decreased. Prontosil was administered. On January 23, new attacks of headache and projectile vomiting occurred. Looking straight ahead nystagmus was present. On January 25, there was very slight nystagmus of first degree to the right; on January 30, the patient felt well. Very slight nystagmus of first degree to the right was present. The caloric test revealed a normal irritability on the right side and a slight hyperirritability on the left side. The patient was dismissed from the hospital.

This patient was seen only 11 days after the onset of the disease. However, from the history it was learned that the girl became sick with a sore throat accompanied by a high temperature and general symptoms such as weariness, drowsiness, and vertigo; later a peritonsillitis on the left side developed. Eight days after the onset of septic angina the first local symptom of the brain, namely, a strabismus, was discovered. When the patient entered the hospital, the peritonsillitis as well as the sepsis had decreased, but we were able to find symptoms which definitely indicated a disease within the posterior fossa. Those symptoms were the following: (1) dysarthria; (2) paresis of the sixth cranial nerve on the right side; (3) ataxia; and (4) spontaneous nystagmus. These symptoms were accompanied by slight meningeal symptoms. As in Case 2, the diagnosis of an encephalitis was made. On account of our experience in Case 2 we must correct this diagnosis and must replace it by the diagnosis of an edema of the brain. We must do that all the more, since all neurological symptoms disappeared after a few days, so that the patient could be dismissed from the hospital.

CONCLUSIONS

Comparing the cases of the 2 groups described, the following can be concluded:

1. In the course of a pharyngogenous sepsis the meninges and the brain can become affected by means of a phlegmon of the parapharyngeal space or by a general sepsis.

2. The infection of the parapharyngeal space can occur without alarming general and local symptoms.

3. The infection of the parapharyngeal space can occur with a critical decrease in temperature and an increase in leucocytes.

4. Neuralgias of the trigeminal nerve can be absent in cases of phlegmon of the parapharyngeal space.

5. The blocking of the eustachian tube in a phlegmon of the parapharyngeal space occurs as a result of infection passing along the connective tissue and blood vessels, which perforate the cartilage of the tube under normal circumstances from the parapharyngeal space into the mucous membrane.

6. At operations for deep abscesses of the neck a normal sheath of the blood vessels should not be incised, when there is a purulent necrosis in the surrounding fascia of the neck.

7. When the infection of the parapharyngeal space reaches the contents of the skull, the meninges are infected first and intradural suppuration and thrombophlebitis of the cavernous sinus and of the carotid plexus develop. All these diseases of the meninges are, as a

rule, accompanied by typical, well known symptoms, but sometimes (Case 1) these typical clinical symptoms are missed.

8. In general sepsis the brain itself is affected first while the meninges are not at all involved or their infection is of secondary importance, which is contrary to infections of the contents of the skull, produced by way of a phlegmon of the parapharyngeal space.

9. The infection of the brain in general sepsis can lead to various pathological entities. In Cases 2 and 3 an edema of the brain was produced.

10. This septic edema of the brain can appear clinically as a disease which is localized within the posterior fossa.

11. Septic edema of the brain can disappear entirely, when the pharyngogenous sepsis is treated in the proper way, and cured.

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FOREIGN BODY ARTHRITIS

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USUALLY when a foreign body enters a joint it is either removed relatively soon after the accident or it carries pyogenic infection into the joint so that a pyogenic arthritis develops. Occasionally, however, a foreign body enters a joint and is permitted to remain in or near the joint over a variable period of time. I believe that most surgeons feel that the continued presence of such a foreign body may injure the joint, but I do not think that much is known about the type or degree of injury which may occur. At any rate, I have not been able to find anything on this subject in the surgical literature. In this paper 6 such instances will be reported in detail.

CASE 1. A girl, 12 years of age, complained of pain, swelling, and disability of the right knee. Seven weeks before the examination a thorn had penetrated the lateral aspect of the knee just above the patella. This had been pulled out, but the next day the knee had been swollen and painful. The swelling had persisted and the pain had subsided with rest, but had returned with use of the extremity. At times there had been some elevation of temperature, but at no time had she been acutely ill.

On physical examination the right knee was moderately swollen, contained a moderate amount of excess fluid and was quite tender, especially over the lateral portion of the joint in the vicinity of the healed puncture wound. It was maintained in a position of slight flexion and movement of the joint was painful and markedly limited. There was some local heat and some thickening of the synovial tissues. The rectal temperature was 101 degrees F. and the white blood cells numbered 10,500. The roentgenogram showed the soft tissue swelling but no evidence of bone change.

The patient was kept in bed for 2 days with the knee immobilized in a large hot wet dressing and the acute symptoms subsided but the swelling persisted. She was then permitted to walk and use the knee, and the acute symptoms re-appeared and operation was advised. The roentgenogram showed soft tissue thickening and slight excess fluid in the joint, but there were no visible changes in the bone (Fig. 1).

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The knee was opened through a lateral incision. When the joint was opened a moderate amount of rather cloudy, straw-colored fluid escaped. The synovial tissue was moderately thickened and hyperemic. Over the lateral surface of the femur just above the cartilage margin there was an area about 1.5 centimeters in diameter where the synovial tissue was especially thickened and elevated above the surrounding surface of the joint. This area was excised and was found to contain a small bit of foreign matter (the tip of the thorn). The hyperplastic synovial tissue was then removed from the lateral portion of the joint and the wound was closed without drainage. The postoperative course was uneventful and the patient obtained an apparently normal knee.

Culture of some of the tissue removed at operation revealed no growth.

Microscopic examination showed marked thickening and increased vascularity of the synovial tissue. The blood vessels were thin walled and the connective tissue was youthful in character, consisted largely of young connective tissue cells, and was diffusely infiltrated with leucocytes and small and medium sized round cells. A few foreign body giant cells were seen in this tissue and some areas contained extravasated red blood cells. The synovial surface was intact and consisted of several layers of living, rather large synovial cells. This surface exhibited rather marked villus formation, the structure of the villi resembling that of the adjacent synovial tissue.

The pathological diagnosis was subacute arthritis, probably infectious in origin.

CASE 2. A man, 65 years of age, complained of pain and stiffness in the left knee. Approximately 45 years ago this patient received a bullet wound in the left knee. The bullet was removed about 3 months later. Apparently it had been embedded in the lateral condyle of the femur. There appeared to have been a low grade infection, as the wound drained for a few weeks after the operation. After the bullet was removed and the drainage had ceased, the knee was very stiff and has remained so. Not only was it stiff, but it was painful in damp weather or after an unusual amount of use.

Physical examination revealed movement in the left knee limited to about 30 degrees and accompanied by rather coarse crepitus. The knee was not tender and there was no excess fluid and no swelling. Roentgenograms showed narrowing of the joint space, irregularity of the femoral condyles, and considerable new bone around the articular margins of all of the bones (Fig. 2). The right knee was normal.

Diagnosis: hypertrophic changes superimposed on an old infectious arthritis.

CASE 3. A woman, 52 years of age, complained of marked stiffness, disability, and moderate pain in the left knee. These symptoms had been of gradual onset over a period of many years. Roentgenograms revealed a foreign body, apparently a piece of a needle about 2 centimeters long, embedded in the soft tissues in the anterolateral portion of the knee. According to the history, this needle was stuck into the knee approximately 25 years ago. The right knee had caused her no trouble.

On physical examination the joint showed marked restriction of motion, marked crepitus on movement, and pain at the limits of movement. There was no excess fluid, but there was moderate peri-articular thickening and moderate tenderness. Roentgenograms showed complete loss of the joint space, marked erosion of the cartilage, and squaring of the condyles of the femur and tibia with a large amount of hypertrophic new bone around the margins of the joint. There was also spur formation and cartilage erosion on the patella. The needle was seen lying in the soft tissues anterior to the joint space in the lateral view and opposite the mesial side of the joint in the anteroposterior view. The right knee showed moderate hypertrophic changes and a joint mouse in the suprapatellar bursa (Figs. 3 and 4).

Arthrotomy was done. The needle was found embedded in the fat pad near the synovial surface. The needle was quite rusted and surrounded by scar tissue. The internal semilunar cartilage was fibrillated. This and some of the hypertrophied fat pad, including the needle, were removed. Also, a considerable amount of new bone around the margins of the articular surfaces of the femur and tibia was removed.

The postoperative course was uneventful, except that the patient was relatively slow in getting back on her feet and the knee is still the site of hypertrophic changes. There is, however, marked improvement in the function of the knee.

Microscopic examination of the bone showed marked degeneration of the cartilage with disintegration and erosion, bare eburnated bone being exposed in some areas. The synovial tissue showed fibrosis, sclerosis of blood vessels, and some villus formation, the villi containing a considerable amount of fibrous tissue. In the sections examined there was no synovial proliferation. However, there was some infiltration in the subsynovial tissues and occasional medium sized foreign body giant cells were seen. These were surrounded by small round cells, but the structure was not typical of the changes caused by tuberculosis.

Diagnosis: hypertrophic arthritis of the knee due to a foreign body.

CASE 4. A young woman, 29 years of age, complained of pain and disability in the left ankle following turning it under her 7 years ago. She stated that the pain had been present since. She had been treated as a psychoneurotic. She then went to a chiropractic college and had a roentgenogram made which showed a loose piece of glass in the ankle joint, which was there diagnosed as a fracture. Further history elicited the fact that 15 years ago a plate glass window fell on the left leg and she suffered a small cut in the calf of the leg which healed promptly and caused no further trouble.

New roentgenograms showed a piece of glass approximately ¾ of an inch long, ¼ of an inch wide, and ⅛ of an inch thick lodged in the posterior portion of the ankle and lying in the space between the margins of the tibia and the fibula. There was definite narrowing of the joint space and also production of new bone around the margins of the ankle joint.

Through a posterior incision the foreign body was removed on October 6, 1935. Recovery was uneventful. Examination of the joint at the time of the operation showed thinning and some complete erosion of the articular cartilage of the superior surface of the astragalus and the inferior surface of the tibia, pigmentation of the synovial membrane in the region of the foreign body, and slight thickening and villus formation in the region of the foreign body. The piece of glass was lying loose in a cavity which was continuous with the synovial cavity and its lower extremity projected into the joint and rested on the superior surface of the astragalus.

Since there was only slight improvement in the patient's symptoms after the operation and since she continued to have pain on movement of the ankle and was not able to walk without support, she re-entered the hospital on December 5, 1935, at which time an arthrodesis of the ankle was performed with a sliding graft from the tibia. She was discharged in a plaster cast and her recovery was uneventful. She is now walking well with a stiff ankle and has no pain.

Diagnosis: degenerative arthritis of the ankle due to a foreign body.

CASE 5. A man, 22 years of age, complained of pain and disability in the left foot. About 4 years ago this patient received a bullet wound in the left foot from a 22 caliber rifle. Apparently the bullet was lodged in the head of the astragalus. The wound healed without complications.

Roentgenograms which the patient brought with him showed a large amount of opaque substance, apparently lead, in the astragaloscaphoid joint with marked narrowing of this joint space. This opaque material outlined the synovial surface of the joint and also extended down into the subastragaloid joint and more or less surrounded and extended into the head of the astragalus (Fig. 6).

At operation the astragaloscaphoid joint was exposed through a median incision. The fragmented remains of the lead bullet were found to be embedded in the cartilaginous surface of the head of the astragalus. The surrounding cartilage was blue and gelatinous in consistency. This merged into areas in which the cartilage was very thin over the bone. The synovial surface was thickened and dark brown in color. As much of the synovial surface as could be reached from this incision was removed.

The remains of the bullet were removed from the head of the astragalus, the defect in the bone was saucerized, and the joint closed without drainage.

The postoperative course was uneventful. However, the patient still has some pain in the foot. This pain is aggravated by use and it may be necessary to arthrodese the joint.

Microscopic examination of the synovial membrane showed rather marked round cell infiltration with increased vascularity and moderate villus formation. In the deeper subsynovial tissues adjacent to the fibrous capsule, masses of amorphous material were seen. This was presumably lead. The articular cartilage showed necrosis and disintegration with some active regeneration as indicated by the presence of large cell nests surrounded by apparently necrotic matrix. The amorphous precipitate referred to was extracellular and encapsulated and in some places formed little globules. The capsule was of connective tissue and numerous foreign body giant cells were seen in the area. It is to be noted that in these areas there was little or no foreign material in the cells. There was considerable cellular débris in the joint and the hyperplastic synovial cells appeared to be migrating into the joint cavity. The masses of precipitate of lead were surrounded by cells and at times by giant cells, but there was very little tendency for the cells to phagocytize these masses. The method of removal appeared to be the inclusion of the precipitate by cells growing around it. There were practically no leucocytes.

Diagnosis: degenerative arthritis due to a foreign body.

Fig. 1. Case 1. Slight soft tissue thickening and slight excess fluid in the joint 7 weeks after a thorn entered the knee. No bone change.

CASE 6. This consists of roentgenograms of a spine which were sent to me by Dr. William K. Ishmael of Oklahoma City. According to the history this patient was injured 12 years ago when a rifle bullet entered the abdomen and lodged in the spine between the second and third lumbar vertebræ. He was severely ill on account of the abdominal injury, but recovered and states that the back has not at any time caused him any trouble. He consulted Dr. Ishmael on account of pain in the heels which was associated with calcaneal spurs. The roentgenograms show the bullet still in its original position while the nucleus pulposus has disappeared and

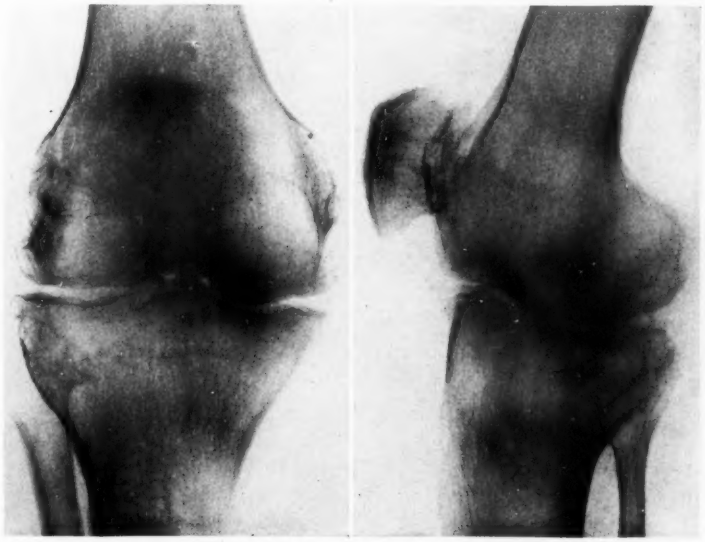

Fig. 2. Case 2. Marked degenerative and hypertrophic changes in the knee joint 45 years after a bullet wound in the lower end of the femur (probably infected).

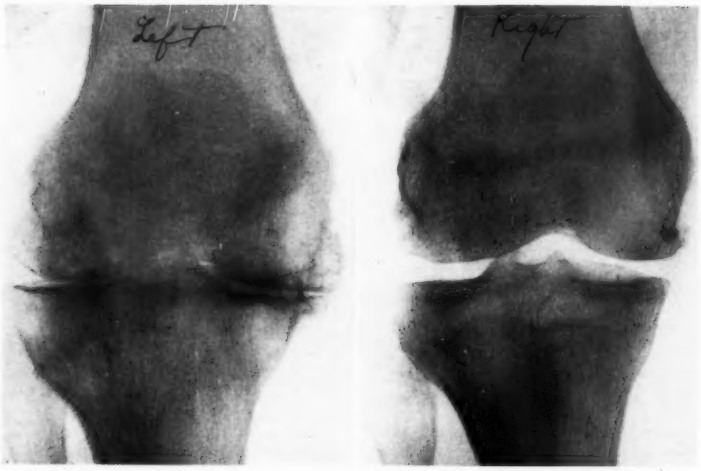

Fig. 3. Case 3. Anteroposterior views of both knees showing marked degeneration and hypertrophic changes in the left knee with a piece of needle at the joint level on the mesial side. This had been in place 25 years.

the bodies of the second and third lumbar vertebræ are fused by bone. There is no visible rarefaction of the bone around the bullet and there are no hypertrophic changes in the adjacent regions of the spine.

Diagnosis: painless fusion of the vertebral bodies due to a lead bullet.

In Cases 1 and 2 it is probable that the foreign body was accompanied by a low grade infection. In the case of the young girl with the point of the thorn in her knee, the knee was at times hot, tender, painful, and the patient had a slight elevation in temperature.

Fig. 4. Same as preceding. Note the marked changes in the left knee and the needle present in the tissues anterior to the upper end of the tibia. There is a small joint mouse in the right knee which caused no symptoms.

However, the culture of the material removed at the operation was negative, the joint was closed without drainage, the wound healed by primary intention, and the patient made an uneventful recovery and obtained an apparently normal knee. It is thus evident that the infection, if present, was of a relatively mild type.

In Case 2 it is not known definitely whether the joint proper was infected or not, but it is probable that it was and that this infection was relieved by removal of the foreign body from the condyle of the femur about 3 months after the injury. This probable infection plus the foreign body remained in the joint long enough, however, to cause marked damage to the joint and resulted in a knee which showed marked degenerative arthritis and limitation of motion with pain after excessive use.

In the 4 other cases there apparently was no infection. The needle remained in the knee joint, or at least in the tissue near the synovial surface of the knee joint, for 25 years and was embedded in scar tissue. In this knee extreme hypertrophic arthritis developed while only moderate hypertrophic changes were present in the other knee. The relatively inert piece of glass in the ankle joint had apparently been there about 7 years, as the patient stated

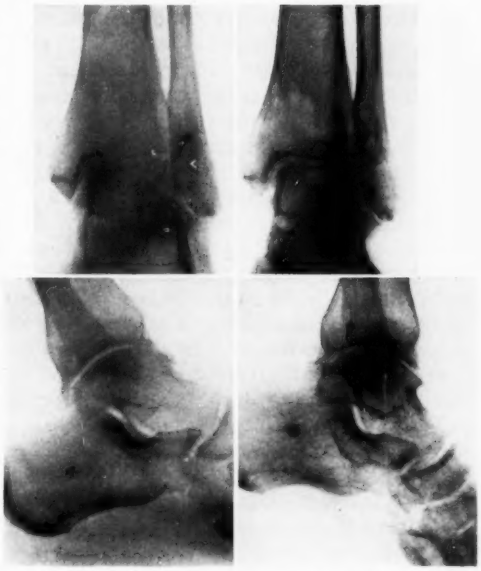

Fig. 5. Case 4. Anteroposterior and lateral views of the ankle before and after operation showing the degeneration and narrowing of the joint surface and some hypertrophic changes around the joint margin. On the left a piece of glass is seen in the lateral and posterior portion of the ankle joint. Later an arthrodesis of the ankle was performed with a sliding graft from the tibia.

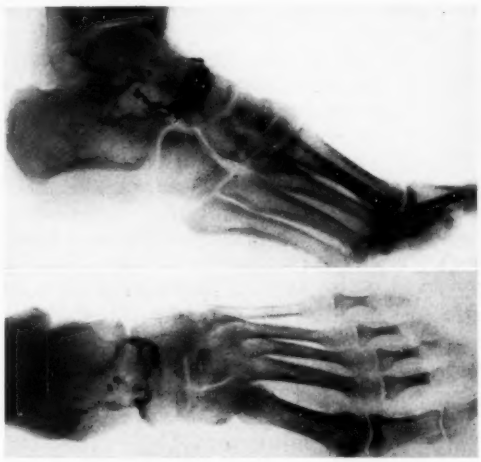

Fig. 6. Case 5. The opaque material is a disintegrating lead bullet in the astragaloscaphoid and subastragaloid joints. Note the narrowing of the joint space.

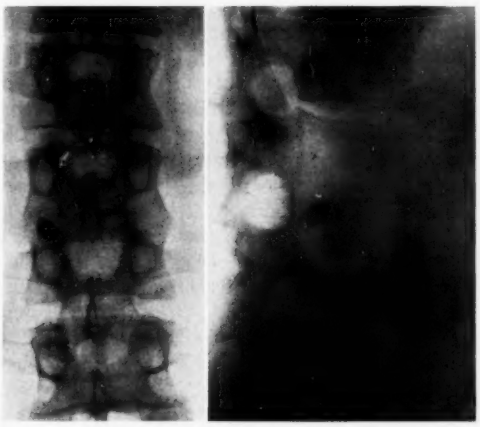

Fig. 7. Case 6. Anteroposterior and lateral views of the lumbar spine. Note the disappearance of the intervertebral disc and fusion of the vertebræ with the lead bullet lying in the bone and apparently causing no reaction around it. There were no symptoms. This is 12 years after the injury. (Case of Dr. W. K. Ishmael.)

that she had had symptoms in that joint for that period. As a result of the irritation there was marked degeneration of cartilage and the development of a hypertrophic arthritis which was so severe that after the glass was removed the joint remained so painful that an arthrodesis of the ankle was necessary in order to relieve the patient's symptoms.

In the case of the bullet which remained projecting into the joint for 4 years, it is to be noted that the bullet was almost completely disintegrated, that minute particles of lead had spread and had been incorporated in the tissues over the entire synovial surface, and that there was marked degeneration of the cartilage over the head of the astragalus and over the scaphoid. This degeneration extended quite far beyond the bullet and had progressed to such a degree that removal of the remains of the bullet, smoothing off of the bone, and removal of most of the synovial tissue resulted in only partial relief and an arthrodesis may be necessary later. The bullet in the intervertebral disc caused degeneration of the disc and painless fusion of the bodies of the vertebræ.

It is thus evident that foreign bodies should not be permitted to remain in or near joint cavities. In some instances they may keep active a mild infection. In instances in which there is no infection they tend to cause degenerative arthritis and this arthritis is apt to progress more rapidly and to result in more destruction of the joint than is found to accompany joint mice, which are pieces of the normal joint structure and may remain in a joint over a period of many years and cause relatively slight changes, although their presence does tend to cause the development of a slowly progressive hypertrophic arthritis.

In the case in which the piece of glass remained resting upon the posterior lateral portion of the superior articular surface of the astragalus the degeneration of cartilage was widespread and it was most marked on the bearing surfaces of the joint. It is thus evident that a foreign body in a joint can cause damage to the articular surface other than by direct mechanical injury. The same is true of the piece of needle which lay adjacent to, but not in the joint cavity.

In the case of the lead bullet in the astragaloscaphoid joint, it is interesting to note that this bullet was almost completely disintegrated and that the particulate material resulting from the disintegration of the bullet appeared to have been removed from the joint cavity by a process of inclusion in growing tissue and not as we would expect, by phagocytosis. In the sections examined no lead was seen in the bodies of any cells, although numerous small foreign body giant cells and macrophages were present. Apparently these cells refused to take up the minute particles of lead. However, there was no extreme fibrous tissue reaction, as masses of material, a large portion of which was found to be lead, were friable and found to be easily cut with a knife.

CONCLUSIONS

1. A foreign body in or near a joint cavity may serve to prolong and keep active a mild low grade infection and result in destruction of the joint.

2. A non-infected foreign body in or near a joint cavity may cause severe progressive degenerative arthritis in the joint.

3. Foreign bodies in or near joints should be removed before the degenerative changes are produced.

4. Foreign bodies in intervertebral discs may cause degeneration of the disc and fusion of the bodies of the vertebræ. Unless they are infected they need not be removed, because the fusion causes no symptoms.

THE EFFECT OF SUBCASTRATIVE ROENTGEN THERAPY ON OVARIAN PHYSIOLOGY

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DISORDERS of ovarian function fall into several large groups of clinical importance. We are concerned primarily with the value of subcastrative amounts of x-rays in the treatment of anovulatory flow, temporary or prolonged amenorrhea, and sterility in the absence of demonstrable male or female causes or when associated with disturbed menstruation. We have discarded for this discussion those cases of ovarian failure with obvious congenital anomalies, or disorders of the ovary recognized as secondary to imbalance of other members of the endocrine system.

An abundance of literature exists concerning the use of various endocrine preparations for the correction of these conditions, but most of the reports are not quite conclusive. Subcastrative roentgen therapy has been presented as a very satisfactory therapeutic agent. The previous work in this field was done before or during the time when the pattern of the endometrium, as it reflected the ovarian sequence of follicular growth, rupture, and luteinization, was being unfolded. Hence the disorders of menstruation were not characterized as ovulatory or anovulatory, nor was the change in ovarian physiology brought about by roentgen therapy demonstrated.

On the basis of extensive biological studies, we may depend on demonstrable progestin effect in the endometrium as indicative of ovulation. The specific cytological changes evoked by progestin are well known (2, 5, 22) and are demonstrated in the accompanying photomicrographs. The term, menstruation, unqualified, has been used loosely for some time, but now it is properly applied only to the flow from such progestational or secretory endometrium. Flowing which is more or less cyclical may come from a proliferating endometrium. Now, when such a flow in periodicity, amount, and duration resembles normal catamenia, it is called anovulatory menstruation.

We accept the theory that ovulation occurs from the sixteenth to the twelfth day, and usually about the fourteenth day before true menstruation. We assume, if biopsy reveals a secretory endometrium, that ovulation has occurred and that menstruation is to be expected within 14 days.

From the Fertility Clinic, Free Hospital for Women, Brookline.

PREVIOUS REPORTS ON ROENTGEN THERAPY

Halberstaedter, in 1905, announced the specific sensitivity of the ovaries to x-ray, and according to Edeiken, Chereon, Duvel, Kronig, Seitz, and Wintz in 1910 to 1911, brought to the attention of roentgenologists the "stimulating" action of the x-rays on the ovary. In 1915, Van de Velde reported the return of normal ovarian function after small doses of x-ray had been applied over the ovaries. Beneficial results were presented often in the literature of the continent after that. As the activity of the anterior pituitary became linked with that of the ovary, similar small amounts of x-rays were given over the pituitary as a "stimulating" dose. Edeiken, in 1933, was one of the first to employ roentgen therapy in the United States. He reported 56 amenorrheic patients of whom 40 had return of normal periods, and 33 sterile patients of whom 14 became pregnant (1 patient twice) with no evidence of any harmful effect. Mazer and Spitz (19), in 1935, reported 102 cases of amenorrhea, oligomenorrhea, metrorrhagia, associated sterility, and primary dysmenorrhea, stating that well over half of the patients were benefited by the treatments. Kaplan (12, 13), Squire and Allen, Leavitt, Bromley and Phillips, Ford and Nelson, re-

ported series and results, good or variable. All emphasized the importance of first ruling out pathological states as a cause of the menstrual disturbance, of eliminating all but the "functional" ovarian disturbances, and the fact that no harmful effects had been noted. The technique of radiation was essentially the same as that employed in this group. Certain case results, in the Mazer and Spitz (19) group were evaluated by a study of the endometrium, but the findings were not stressed. This we have endeavored to do in our series.

EFFECT OF ROENTGEN THERAPY ON THE OVARIES AND PITUITARY

The general action of the x-rays has been deduced from their effect upon plant and animal life and upon clinical observations. Edeiken points out that the theories brought forth to explain the action of x-rays can be divided into 2 modes: (1) stimulation and (2) selective destructive action. The Arndt-Schulz law applied to the biological effects of the x-ray, states that small doses of x-ray stimulate, moderate doses inhibit, and large doses destroy vital processes. We are able to establish the upper limit, that of destructive action of the x-rays, since the sterilizing dose for the human female at various age levels is known. Whether the small dosages employed therapeutically in these cases would stimulate or inhibit is open to conjecture. Those who support the theory of indirect stimulation of the ovary report a few cases in which there was resumption of normal ovarian activity after roentgen therapy had been employed on the pituitary alone. It is additionally suggested that minimal exposure of the ovaries effects the destruction of a persisting follicle with its deranged estrogen production and thus allows a normal follicle to begin its maturation. Traditionally, a "persisting corpus luteum" has been blamed for such failure of ovulation. But corpora lutea are notably resistant to x-ray. Furthermore, as a result of endometrial study, we believe that a "persisting corpus luteum" which produces progestin sufficient to inhibit ovulation, is extremely unlikely, if it occurs at all, save only in the early months of pregnancy. To us then, the most attractive theory brought forth to explain good roentgen results is: A persisting follicle with its troublesome endocrine activity is destroyed and new follicles allowed to mature and to re-establish normal cycles.

Mazer and Spitz (19) for 3 years followed 11 normally menstruating women who had received this small dose of x-rays to both pituitary and ovaries. In no single case was the menstrual rhythm seriously or permanently affected. However, all of the writers note a very low incidence (2 to 5 per cent) of temporary amenorrhea with or without hot flushes, nervousness, and the like, suggestive of decreased or absent ovarian function. Whether this would have happened in these patients anyway, or whether it was due to roentgen therapy is difficult to say, but it does serve as a warning, because older patients, whose ovaries have lost the vigor of youth, may be less tolerant of this treatment. This transient disturbance will be shown in a considerably higher percentage of our cases than is usually recorded. All other writers emphasize the absence of subsequent obstetrical disasters from fertilization of defective ova. In our series of cases there was 1 miscarriage in the early months of gestation. We cannot identify the cause. However, should an abnormal ovum become fertilized, it is unlikely that it would be carried to term (10).

We make bold to suggest that there is little value in x-ray treatment of the pituitary, either alone or in conjunction with the ovaries. Hartman and Smith, in 1938, reported a study of 13 non-ovulating rhesus monkeys who were given a single dose of between 60 and 400 r's to the pituitary. There was no increase in ovarian size in 11 animals and in only 1 case did the animal ovulate. This is not unusual even without treatment. There were no harmful sequelæ, for in one-third of the monkeys spontaneous ovulation and conception occurred in the following breeding season.

X-RAY TECHNIQUE[1]

"The factors employed were 200 kilovolts, 50 centimeters focal skin distance, 0.75 millimeter copper equivalent filter. When the pituitary was

[1]We are deeply indebted to Dr. Richard Dresser for treating our patients and for this description of technique.

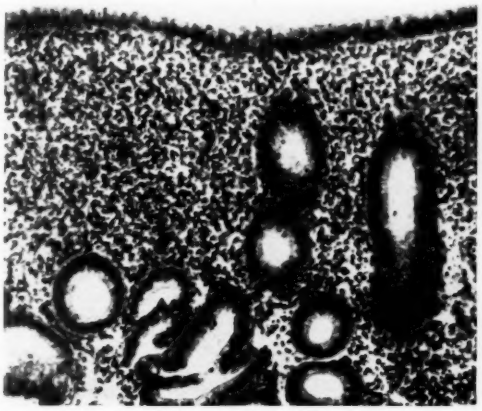

Fig. 1. Case 2, before treatment. Proliferative endometrium, early. Simple glands moderately dilated. Glandular epithelium is columnar with beginning pseudostratification and frequent mitoses. Stroma nuclei are small, closely packed, chromophilic, and are almost free of cytoplasm.

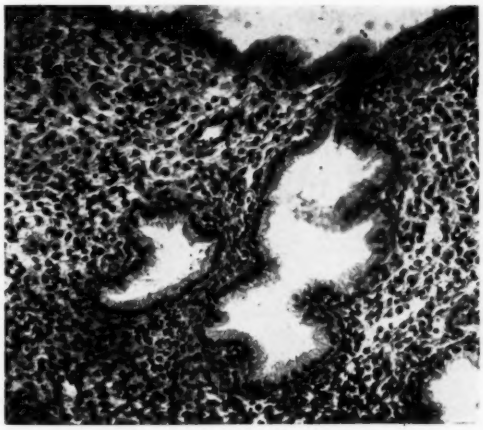

Fig 2. Case 2, after treatment. Secretory endometrium, twenty-seventh day. Serrated glandular epithelium with basal nuclei, fraying, and vacuolization of cytoplasm. Prominent arterioles with thickening of surrounding stroma. Cytoplasm of stroma cells has increased.

treated, the ovaries and pituitary were irradiated at the same sitting. Treatment was given at weekly intervals for three or four weeks. In the average sized woman, a portal of 100 square centimeters was employed over the anterior pelvis, the lower border of the field being centered just above the symphysis pubis. The pituitary was irradiated through a lateral portal of 80 square centimeters. An exposure of 50 r's, air measurement, was given to each area.

"One may assume that the ovaries lie at a depth of eight centimeters from the anterior surface of the pelvis and that the pituitary is seven to eight centimeters distant from the lateral surface of the skull. Ionization measurements made in a pressed wood phantom give a surface dose, with back scattering of 66 r's. Twenty-seven r's were delivered at a depth of eight centimeters, thus dissipating over half the dose to gain sufficient penetration.

"The total dose to the ovaries, given in the four week period was therefore 108 r's, or 81 r's in the three week period. A similar dose, perhaps slightly less, due to differences in conditions of back scattering, was delivered to the pituitary."

Both the ovaries and pituitary were irradiated in the majority of cases, the ovaries alone in several, but in no case was the pituitary alone treated. Some of the patients received a duplicate series of treatments after an interval of 2 or 3 months. It has been found that no injurious effects result if such an interval elapses between treatments. Pregnancy should always be ruled out before a second series is started.

EFFECT OF INSTRUMENTATION, HORMONES, RADIUM

Consideration must be given to other procedures or adjunctive treatments which were employed while attempting the relief of these patients. It is a recognized fact that in cases of endocrine imbalance of this type any form of cervical instrumentation may be sufficient to tip the hormonal balance to normal. Frequently, normal ovulatory cycles succeed previous irregularity, or pregnancy occurs following a period of infertility, after a simple dilatation of the internal os, a curettage, a cauterization of the cervix, or after an endometrial biopsy. In one of the patients not included in this series, 4 endometrial biopsies were taken at weekly intervals before the patient could arrange for x-ray treatment, after which, ovulation promptly began. Endometrial biopsy was necessarily involved in the study of all our patients, but as the large majority had been subjected to such a procedure unsuccessfully for months or even years before roentgen therapy was employed, this factor is presumably minimal.

All specimens were obtained by the small suction curette according to the methods described by Klingler and Burch, Rock (21), Novak, Kurzrok, Rock and Bartlett (22), and

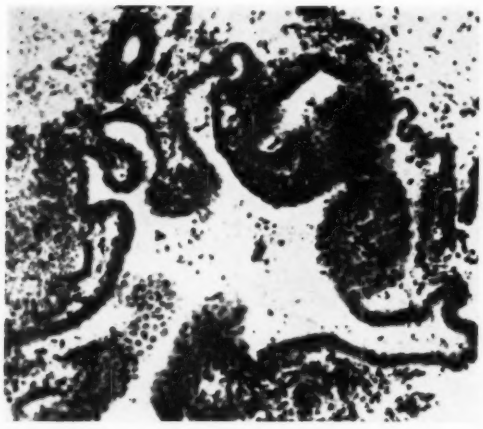

Fig. 3. Case 6, before treatment. Hypoplastic endometrium. Simple glands. Infrequent mitoses. Thin stroma with small nuclei.

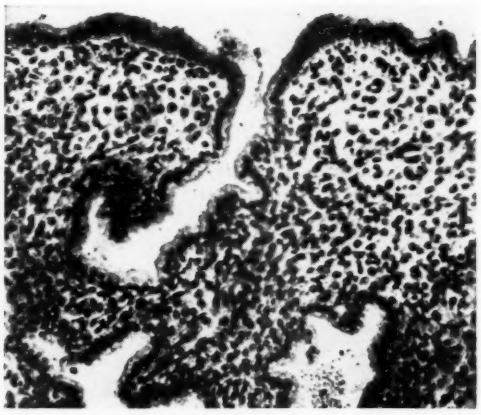

Fig. 4. Case 6, after treatment. Secretory endometrium, twenty-sixth day. Frayed edges of glandular epithelium with basal nuclei. Absence of mitoses. Large stroma nuclei with increasing cytoplasm and diminishing edema.

others. The biopsies were taken as an office procedure without anesthesia or with intravenous injection of 2 to 3 cubic centimeters of evipal soluble as an analgesic. No harmful effects have been observed in hundreds of procedures. Specimens were obtained from high on either the anterior or posterior wall of the fundus uteri, for here the endometrium tends to show the cyclical progression of events best in either the normal or abnormal cycle. Biopsies were taken during the phase of possible corpus luteum activity, i.e., from 2 to 16 days before the expected onset of the next period. Failure to find secretory activity of the endometrium during this time was regarded as evidence that ovulation had not taken place and that any subsequent flowing within 10 days was anovulatory.

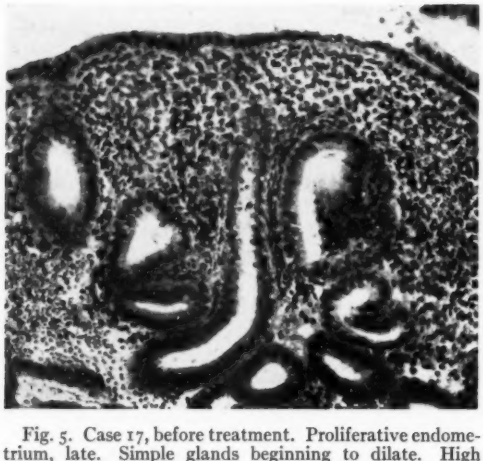

Fig. 5. Case 17, before treatment. Proliferative endometrium, late. Simple glands beginning to dilate. High columnar epithelium with pseudostratification and frequent mitoses. Densely packed stroma with nuclei. Extravasation of erythrocytes.

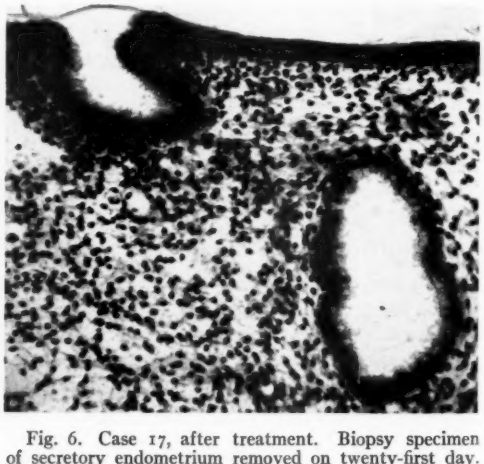

Fig. 6. Case 17, after treatment. Biopsy specimen of secretory endometrium removed on twenty-first day. Uniformly basal glandular nuclei with vacuolization of cytoplasm and absence of lucid zone. Marked generalized edema of stroma.

An appreciable number of these patients was given various hormone extracts before the x-ray treatment. In only 1 patient (Case 10) did they play a confusing part. All cases in which the endocrines totally obscured the picture were discarded.

Since x-ray and radium have analogous effects upon the ovaries, it is reasonable to substitute equivalent doses of radium for x-ray. In 2 patients of this series (Cases 12 and 13) radium was employed during the course of treatment. These are indicated in the clinical records. One of these patients responded well to treatment (Case 12), the other did not (Case 13).

SELECTION OF CASES

Forty patients were selected from the endocrine clinic of the Free Hospital for Women and from this group we have chosen 27 whose records we are able to present in full. These cases may be classified as follows:

1. *Anovulatory flowing with sterility.* There are 10 cases in this group. As will be indicated in the clinical record, some of these patients had an occasional ovulatory cycle as shown by biopsy, but habitually failed to ovulate. In most cases sterility was the presenting complaint. Anovulatory menstruation has been defined here as cyclical flowing, usually without a constant interval, from a proliferative endometrium. This is commonly considered due to persistent follicles which secrete estrogen. The picture of proliferation is shown in the photomicrographs. In the instances of scanty or rare flow, we have purposely not used the term, "oligomenorrhea," which is applicable we think, only to infrequent normal, or ovulatory menstruation.

2. *Metrorrhagia.* This we interpret to mean periods of flow which vary widely in interval, duration, and amount. We present 5 cases in this group, in 4 of which flowing was from a proliferative endometrium and in only 1 (Case 13) from a secretory endometrium. This may have been a chance finding in a patient who habitually failed to ovulate. It may be an instance of a deranged bleeding mechanism.

3. *Amenorrhea.* This we define as the total absence of catamenia for not less than 3 months. Six cases of amenorrhea with associated sterility and 1 case of amenorrhea in an unmarried patient are presented. The intervals between periods varied from 3 months to 8 years. Three of these patients had had previous pregnancies.

4. *Sterility with normal ovulatory cycle.* Two cases of apparently normal menstruation are described in which all other demonstrable factors of sterility had been excluded before roentgen therapy was employed.

5. *Hypermenorrhea.* Two cases with normal ovulation are presented in which bleeding occurred which required more than 6 napkins a day for more than 5 days.

6. *Polymenorrhea.* One case of regular ovulatory flow is presented in which cycles were habitually shorter than 24 days.

These groups include the types of cases which, according to reports, are benefited by roentgen therapy, with the exception of primary dysmenorrhea. We have treated several cases of primary dysmenorrhea with results which cannot be evaluated because of inadequate biopsy studies. It may be noted, however, that they failed to respond symptomatically.

ANOVULATORY FLOWING WITH STERILITY

CASE 1. Mrs. B., FHW 123/36, a 32 year old Russian-born housewife, complained of sterility for 12 years. Catamenia were scanty and irregular, occurring two or three times a year before marriage. From the time of marriage they had become more frequent and less irregular, on a 25 to 35 day cycle, lasting for 3 days and requiring 2 pads a day. Repeated biopsies of the endometrium during the expected secretory phase, as well as just before the x-ray treatment, failed to show evidence of ovulation. No other cause for sterility was found

A dosage of 180 r's over the ovaries alone was given. Four and one-half weeks after this treatment a 6 day flow, requiring 5 to 6 pads a day, commenced. A biopsy taken during the secretory phase of the next expected catamenia showed a 17 day secretory endometrium, and on the twenty-seventh day a 25 day secretory endometrium. Flow commenced on the thirtieth day. Repeated endometrial specimens have shown continued normal ovulatory cycles at 28 to 32 day intervals. No further treatment has been necessary. The patient has not conceived.

CASE 2. Mrs. S., FHW 104/398, a 26 year old American housewife, complained of sterility for 1 year. Catamenia were grossly irregular, from 1 to 3 months apart, until a dilatation of the cervix and curettage were performed 6 months before entry to the clinic for a questionable incomplete miscarriage.

Since that time periods occurred at about 30 day intervals. This patient was treated for 3 years with hormones, but the endometrium showed only rare ovulatory cycles. An endometrial specimen on day flow started, just before x-ray treatment was given, showed early proliferation and hypoplasia (Fig. 1).

A dosage of 180 r's was given over the ovaries alone. Three weeks after completion of the treatment, it was thought the patient was pregnant, but an Aschheim-Zondek test was negative. Biopsy was withheld for the next 2 months on suspicion of pregnancy, although the patient flowed at 29 to 31 day intervals during that time. Biopsy was taken during the expected secretory phase of the third subsequent period. This showed a 27 day secretory endometrium (Fig. 2). Repeated biopsies over a period of 5 months continued to show evidence of normal cyclical ovulation, with the interval about as before, 28 to 33 days, with flow lasting 5 days and the quantity as before treatment.

CASE 3. Mrs. K., FHW 127/296, a 22 year old American housewife, complained of sterility for 9 months. Catamenia were regular at 35 to 38 day intervals, with 1 to 3 days of flow, requiring a napkin a day. Biopsies throughout the next year showed both anovulatory and occasional ovulatory flowing 1 to 3 times a year. There was no regularity of ovulation, and the patient did not become pregnant.

A dosage of 360 r's was given over both ovaries and the pituitary. Catamenia began 1 month after treatment and recurred every 31 days until she became pregnant 3 months after treatment. The patient returned to the clinic after the birth of the child and was followed for the 3 ensuing years. She had had no coitus since this pregnancy and biopsies were not taken to prove constant ovulation. Catamenia, however, occurred regularly every 30 to 31 days. We consider the patient cured inasmuch as a normal pregnancy began 3 months after treatment and had not been achieved in the 2 years previous to the treatment.

CASE 4. Mrs. G., FHW 25/139, a 30 year old American housewife, complained of infrequent catamenia. She had been married for 12 years and had 2 children, aged 11 and 5. Her periods had been normal until after her first pregnancy. Amenorrhea was constant during the 12 months in which she nursed the child, and for the next 2 years flowing occurred occasionally. Amenorrhea again returned for 4 years. She become pregnant at the end of this period and during the 11 months in which she nursed this child she did not have a normal period. Flowing began in the twelfth month and was repeated only 2 or 3 times a year in the 4 years prior to her admission to the clinic. During this time there was a gain in weight, and hirsutism was noted, but there was no striking abnormality. The basal metabolic rate was plus 5 and plus 9, and no evidence of endocrine disturbance was found. In the next 5 months there were only 3 episodes of flowing, and repeated biopsies showed early proliferation with an 8 day endometrium. Hormone therapy was without result.

A dosage of 520 r's was given over the ovaries and pituitary. Four weeks after the treatment was begun, a curettage for incomplete abortion of a pregnancy older than 1 month was necessary. The patient has refused further treatment and since then has menstruated only 3 or 4 times a year.

This is another case in which the flowing is predominantly anovulatory. Evidently the patient became pregnant in a rare ovulatory cycle and aborted as a result of the x-ray treatment. We consider this case a failure and cannot evaluate the damage to early pregnancy by x-ray dosages of this size.

CASE 5. Mrs. S., FHW 146/305, a 32 year old Russian-born housewife, complained of sterility for 12 years. Menstrual cycles varied in length from 21 to 35 days; flow lasted for 5 days and required 4 napkins a day. Endometrial biopsies taken during a year and a half showed anovulatory, with occasional ovulatory cycles.

A dosage of 400 r's was given over the ovaries and pituitary. An examination, 7 weeks after treatment was completed, showed a pregnancy of 6 weeks which has come to term. No biopsy was taken.

CASE 6. Mrs. G., FHW 131/356, a 21 year old American housewife, complained of sterility for 4 years after an abortion in the early months of marriage. Although catamenia had been normal until 2 years before admission to the clinic, they had become irregular, occurring every 1 to 3 months thereafter, lasting for 4 days and requiring 6 pads a day. In the first 6 months of study there were only 3 episodes of flowing and repeated endometrial biopsies showed hypoplasia (Fig. 3) or early proliferation.

A dosage of 360 r's was given over the ovaries and pituitary. There was no evidence of change and 2 months later the treatment was repeated. A biopsy 1 month after the second treatment showed atrophy of the endometrium, but the patient had no symptoms of destroyed ovarian function. Flow commenced 10 weeks after the second treatment and a biopsy taken a day before the next period, 4 months later, showed a 26 day secretory endometrium (Fig. 4). During the next 3 years repeated biopsies showed the 28 to 31 day cycles to be ovulatory. The patient, however, did not become pregnant.

CASE 7. Mrs. R., FHW 101/316, a 28 year old American housewife, complained of sterility for 2 years. Catamenia were irregular every 1 to 3 months, lasting 5 to 6 days and requiring 5 or 6 napkins a day. Biopsies of endometrium showed proliferation of 8 or 9 days; no evidence of ovulation.

A dosage of 180 r's was given over the ovaries alone. Catamenia began 5 days later and has continued in 28 to 30 day cycles for the last 8 months. We do not have an endometrial biopsy to confirm ovulation, but on the basis of her regular cycle, plus molimina of menstruation, we arbitrarily classify this case as a cure.

CASE 8. Mrs. C., FHW 125/33, a 19 year old American housewife, complained of sterility for 6 months. Until marriage, catamenia had occurred regularly every 28 days, but since then, only once every 2 months. A biopsy taken before treatment showed only endometrial proliferation.

A dosage of 360 r's was given over the ovaries and pituitary. Four weeks later a biopsy showed an 18 day secretory endometrium. Flow commenced 6 days after this biopsy and cyclical periods have been regular each month since then. The patient has not yet conceived.

CASE 9. Mrs. H., 135/5, a 36 year old American housewife, complained of sterility for 8 years. There had been a 7½ months' miscarriage 15 years before and catamenia had been irregular every 2 or 3 months for 11 years. An endometrial specimen showed only hypoplasia of the endometrium.

A dosage of 360 r's was given over the ovaries and pituitary. A specimen taken at the completion of this treatment showed only hypoplasia. Catamenia became steadily less frequent. Three years after treatment the patient returned complaining of hot flushes and nervousness and was obviously entering menopause. It is unlikely that this early menopause can be attributed to her x-ray treatment, for 3 years had intervened. From the catamenial history, it would appear that there had been deficient ovarian function for some years. Roentgen therapy did not help and early menopause came 3 years later. Results from x-ray treatment may be expected certainly within a few months of the treatment.

CASE 10. Mrs. C., FHW 122/276, a 33 year old American housewife, complained of sterility since the birth of her only child 8 years before admission to the clinic. Following this pregnancy, her catamenia which had previously been normal, became irregular with lengthening intervals of 4 or 6 weeks. Repeated endometrial biopsies showed hypoplasia or early proliferation of the endometrium.

A dosage of 180 r's was given over the ovaries. Catamenia remained unchanged and repeated biopsies continued to show hypoplasia of the endometrium. There have been no subsequent pregnancies and the patient would merit another course of treatment.

METRORRHAGIA

CASE 11. Miss S., FHW 135/149, a 13 year old school girl, complained of menstrual difficulties since the age of 7 when she stained for a few days. She received treatment for this condition at the Children's Hospital and did not flow again until at 11 years of age. At this time cyclical flowing began and occurred at intervals of 28 days, lasting 6 or 7 days and requiring 2 or 3 napkins a day. Six months before entry to the clinic, the periods became markedly irregular, coming 1 to 5 weeks apart, with a heavy flow of about twice the previous amount. Just before entry, catamenia had continued without interruption for a month. No endometrial biopsy was obtained, but examination revealed no other abnormalities.

A dosage of 360 r's was given over the ovaries and pituitary. Flowing, which had been continuous for nearly 2½ months, stopped 1 month after the first x-ray treatment. A cycle, which was at first irregular, has occurred every 28 to 32 days for the last 2 years. Development has been normal. No endometrial biopsy was taken at any time. We have arbitrarily called this case cured in view of the dramatic change in the quality of the catamenia and the regularity of the cycles which have continued for the 3 years following x-ray treatment.

CASE 12. Miss T., FHW 120/237, an 18 year old school girl, complained that for 3 years flow had increased in duration and had become a daily occurrence for 3 months before entry to the clinic. Biopsies showed a proliferative phase only. Endocrine therapy was without effect and at the end of a year a dilatation and curettage were performed and 350 milligram hours of radium was administered through the uterine cavity. For the next year, menstruation was grossly irregular in amount, duration, and interval; the cycles varied from 2 to 5 weeks.

A dosage of 360 r's was then given over the ovaries and pituitary. A 6 day flow began 2 weeks after the first treatment and was repeated for the next year in regular cycles of from 25 to 35 days, skipping about 1 month in 4 or 5. There are no biopsies for this period.

It is debatable if this same result could not have been obtained had a second small dose of radium been given or had x-rays been used for both treatments. Actually, the same result is effected with either agent, and its selection is a matter of convenience. We have considered this case cured in view of the established cycle.

CASE 13. Mrs. W., FHW 118/180, a 24 year old American housewife, complained of severe hemorrhage with periods for 2½ years following the birth of her only child. Catamenia lasted for 10 days, and exsanguination was so severe that it caused symptoms of blood loss. The patient was given 1400 milligram hours of radium after a biopsy which showed early proliferation. One week after the radium was given, flow ceased, and did not recur for 2 months when a regular 28 day cycle, lasting for 5 months, commenced. At the end of this period constant flowing again began.

A dosage of 360 r's was given over the ovaries and pituitary, and although the flowing ceased 1 week after the first treatment of this series, it started again 2 weeks later. Inasmuch as the bleeding time was found to be moderately elevated, and the patient was again anemic, a hysterectomy was performed and one ovary removed. Pathologically, nothing was found in either ovary. It is difficult to account for this picture except on the basis of the patient's elevated bleeding time which suggested a purpura-like state.

CASE 14. Mrs. C., FHW 136/166, a 39 year old American housewife, complained of sterility for 3½ years. Catamenia had been regular until 3 months before entry when her flowing gradually became a daily occurrence. A biopsy showed early proliferation with a 10 day endometrium.

A dosage of 360 r's was given over the ovaries and pituitary. Flow ceased almost immediately after the first treatment. Three days after the last treatment, the patient noted hot flushes, nervousness, and irritability which continued for a month. However, flowing began again about 6 days after the last treatment and persisted at intervals throughout the next 6 months. For destruction of all ovarian activity 1,500 r's were then given over both ovaries.

That there is danger in roentgen therapy for patients who are elderly in reproductive life, may again be emphasized. The fact that the hot flushes were temporary and cleared up within a month suggests that the condition was due to her treatment, and not to a co-incidental diminution of ovarian activity.

CASE 15. Mrs. C., 135/166, a 22 year old American housewife, complained of sterility for 14 months. Catamenia had always been regular until the last 3 years, when the flow lasted from 10 days to an entire month and required 1 to 2 napkins a day. Biopsies showed early and late proliferation only.

A dosage of 360 r's was given over the ovaries and pituitary. Further biopsies showed no change and catamenia continued as before, until 3 years after the first x-ray treatment when a second series of 360 r's were given over the ovaries and pituitary.

Biopsies taken after this treatment revealed no improvement in the endometrium nor any evidence of ovulation. Roentgen therapy at 3 or 6 month intervals for a longer period is indicated in this case.

AMENORRHEA WITH STERILITY

CASE 16. Mrs. S., FHW 127/183, a 23 year old American housewife, complained of sterility for 4 years. Catamenia had always been irregular, coming at 1 and 2 month intervals, but had not occurred for 5 months before entry to the clinic. An endometrial biopsy showed atrophy of the endometrium and on one occasion showed 10 day proliferation. Weekly endometrial biopsies failed to induce ovulation and, after 1 year of treatment, the patient was referred for roentgen therapy.

A dosage of 360 r's was given over the ovaries and pituitary. A few weeks after completion of this treatment 28 to 30 day cycles began and the patient became pregnant in 8 months. Inasmuch as the pregnancy proved ovulation, we have considered this case cured by roentgen therapy.

CASE 17. Mrs. D., FHW 151/17, a 31 year old Hungarian housewife, complained of sterility for 8 years. Catamenia had been regular until the birth of her only child 8 years before, but since then had occurred at 4 month intervals. Repeated biopsies showed only early and late proliferation (Fig. 5).

A dosage of 360 r's was given over the ovaries and pituitary. The patient flowed 6 weeks later and an endometrial biopsy taken 6 days before that flow showed a 21 day secretory endometrium (Fig. 6). At the end of 5 months, during which time catamenia occurred regularly every 4 weeks, lasted 5 or 6 days and required 3 or 4 pads a day, amenorrhea again set in. We were able to demonstrate at least one ovulatory cycle ending with the first flow after roentgen therapy. There may have been infrequent ovulation in the past, but for a year before x-ray treatment we were unable to demonstrate a secretory endometrium. We have considered this case as cured by definition, though a relapse occurred.

CASE 18. Mrs. L., FHW 134/263, a 28 year old American housewife, complained of sterility. There was a history of 1 normal gestation 7 years before and 2 miscarriages, the last, 5 years before entry to the clinic. Catamenia were regular at 28 to 30 day intervals until 3 months before entry, when they ceased completely. A biopsy showed a 5 day proliferation.

A dosage of 360 r's was given over the ovaries and pituitary. Flow lasting 4 days began 10 days after the first x-ray treatment and continued in 28 to 30 day cycles for 4 months. Amenorrhea then recurred and lasted for 8 months. No biopsy was taken after treatment. Patient did not wish further treatment.

CASE 19. Mrs. A., FHW 112/123, a 27 year old American housewife, complained of sterility for 2 years. Catamenia had always been infrequent, having occurred no more than 10 or 12 times in all and not for 1½ years preceding entry into the clinic. Endocrine therapy was employed without result for 2 years and biopsies showed proliferation in all stages, but never a secretory phase.

A dosage of 180 r's was given over the ovaries. A biopsy then showed atrophy of the endometrium. There was no flowing of any sort. Five months later 360 r's over the ovaries and pituitary was given. A biopsy again showed atrophy and there was no flowing. Under continued endocrine treatment with antophysin and prephysin 1 secretory phase was shown and 1 year later the patient became pregnant. A normal infant was delivered at term.

This case cannot be considered cured by roentgen therapy since pregnancy followed endocrine therapy. One may point out, however, that 2 series of x-ray treatment to the ovaries and 1 to the pituitary had no effect upon the child.

CASE 20. Mrs. D., FHW 132/354, a 27 year old American housewife, complained of sterility and amenorrhea which had been present for the 3 years following the birth of the youngest of her 2 children. The periods had previously been regular. Repeated endometrial biopsies showed early proliferation.

Following some staining which was thought at first to be due to endocrine therapy, 1 biopsy showed necrotic decidua. The patient had surreptitiously miscarried, though there had been no evidence of ovulation during 3 years. After the miscarriage, there was no flowing for 2 months, and biopsy showed only proliferative endometrium.

A dosage of 350 r's was then administered over the ovaries and pituitary. Five weeks after treatment was started, biopsy indicated ovulation. The patient has been followed for 3 years, with frequent biopsies, and has continued to have regular ovulatory menstruation since. She was one who, before treatment, ovulated at long intervals and became pregnant during one of those cycles. Three months after a prompt abortion roentgen therapy was employed and the patient began regular ovulatory cycles which have continued for at least 3 years.

CASE 21. Mrs. W., FHW 15/350, a 28 year old American housewife, complained of sterility for 10 years. Catamenia, previously scanty and infrequent, had been absent for 5 years before entry to the clinic. Biopsies showed only hypoplasia of the endometrium. A dosage of 360 r's was given over the ovaries and pituitary. Biopsies of the endometrium after treatment showed no change in the cellular pattern. This case is considered a failure.

CASE 22. Miss L., FHW 133/388, a 25 year old Italian seamstress, complained of never having had a period. There was no other evidence of endocrine dystrophy. No mucosa was obtained by repeated biopsies. Two courses of 360 r's, 3 years apart, were given over the ovaries and pituitary with no change in the picture.

STERILITY WITH NORMAL MENSTRUAL CYCLE

CASE 23. Mrs. B., FHW 126/111, a 35 year old Canadian housewife, had been barren during 13 years of marriage. Catamenia were regular with a 30 day cycle, lasted 2 or 3 days and required 3 or 4 napkins a day. Repeated endometrial biopsies demonstrated progestin effect, indicating that the patient had true ovulatory cycles with menstruation.

A dosage of 360 r's was given over the ovaries and pituitary. After treatment, given *fault de mieux*, as all demonstrable fertility factors were present, the patient developed amenorrhea with hot flushes for 7 months. She then resumed her regular cycles with ovulation, again proved by biopsy. She has never been impregnated.

CASE 24. Mrs. M., FHW 130/174, a 21 year old American housewife, complained of sterility after 1½ years of marriage. Catamenia occurred every 28 or 30 days, endured for 5 days and required 5 napkins a day. Endometrial biopsy proved that these were normal ovulatory cycles. Male, as well as other female factors being found to be normal, 360 r's was given over the ovaries and pituitary, for experiment. Subsequent biopsy showed no change in the endometrial picture. The patient has not become pregnant in the 3 subsequent years.

HYPERMENORRHEA

CASE 25. Mrs. C., FHW 146/355, a 31 year old Dutch housewife, complained of sterility during 8½ years of marriage. Catamenia were regular at 25 to 32 day intervals, with profuse flow, clots, and some pain. Endometrial biopsy showed questionable secretory activity. A dosage of 150 r's was given over ovaries without change in clinical picture.

CASE 26. Miss H., FHW 135/248, a 37 year old single factory worker, complained of increasingly frequent and profuse periods. Seven months prior to entry, flow had occurred every 18 days. Endometrial biopsies showed normal ovulatory cycles.

A dosage of 600 r's was given over the ovaries and pituitary. Treatment was followed by amenorrhea and hot flushes for 5 months, following which, hypermenorrhea recurred and required hysterectomy. No ovarian pathology was found.

POLYMENORRHEA

CASE 27. Mrs. M., FHW 126/297, a 38 year old American housewife, complained of periods which came every 18 or 19 days and required 3 pads daily. There had been 2 normal pregnancies during the last 7 years before entry. Endometrial biopsy showed regular ovulation.

A dosage of 360 r's was given over both ovaries and pituitary. There was no subsequent change in the endometrial picture, yet 2 months after the treatments, the patient began to complain of hot flushes and nervousness which have persisted for over 2 years without change.

Again we have a patient in the older age group who has suffered from a probable decrease in ovarian activity following roentgen therapy in these dosages.

RESULTS

In all, 27 cases have been reviewed. Twenty-two of these cases were of proved anovulatory flow or of flow which was predominantly anovulatory with occasional ovulatory cycles. One case, in which no endometrium was obtained on repeated efforts, is presumed to be an instance of primary pituitary deficiency. There were 5 cases of cyclical ovulation, but with associated disturbances for which roentgen therapy was employed. These last cases fell into the group which had previously been reported by the radiologists as successfully treated. The cases here reviewed fall into two groups: The first is that with absence or frequent failure of ovulation. There were 22 such cases. The second group of 5 patients includes those with normal ovulatory cycles

but with the associated disturbances of sterility, polymenorrhea, or hypermenorrhea.

TABLE I.—RESULTS OF ROENTGEN THERAPY

Type of case	No. of cases	Cured		Failed		Pregnant
		No.	Per cent	No.	Per cent	
Absence or frequent failure of ovulation	22	12	54.5	10	45.5	3
Normal ovulatory cycles but with associated sterility or abnormalities of menstruation	5	0		5		0

Of the 5 patients with normal ovulatory cycles but associated complaints, none was relieved or cured. But among the patients in group I in whom ovulation was faulty, the percentage of relief or cure was good. Of these 22 patients, 12 were cured, and 10 were failures.

We have necessarily chosen arbitrary standards of relief, cure, or failure. Failures are those in which we could not prove an ovulatory cycle within 3 months after the completion of roentgen therapy. Cured patients are those in whom biopsies demonstrated ovulatory cycles 3 months or more after roentgen therapy, or those of sterility in which pregnancy proved ovulation, or those who acquired after treatment the molimina of menstruation with periodicity of characteristic flow.

Twelve patients of the 22 treated with x-rays, of predominantly anovulatory flowing, satisfy our criteria of cure. The percentage of cure is 54.5, and that of failure is 45.5. Three of the patients became pregnant, or a percentage of 13.6. These figures of cure are well above the most optimistic results reported from endocrine treatment alone. In fact, an appreciable number of these cases had been through the accepted endocrine therapeusis before roentgen therapy was tried.

ANALYSIS OF STUDY

As was previously reported from our clinic (23), occasional anovulatory periods were detected in 9.1 per cent of 392 thoroughly studied cases of sterility. In 4 per cent of these there was evidence of habitual anovulatory menstruation. Mazer, Israel, and Kacher (18) found that 30 per cent of their patients with functional sterility failed to ovulate. Various others report an incidence of from 20 to 50 per cent. In other words, this is not an uncommon condition in women whose sterility is otherwise inexplicable. Added to the distress of disturbed menstruation, such a frequency emphasizes the importance of finding some means to induce ovulation.

We believe that small doses of x-ray are not only not harmful, but that they also offer an appreciable promise of relief. This occurred in 54.5 per cent of our cases.

We do not postulate that any new or peculiar effect is produced by the x-ray when applied over the ovaries or the pituitary. Our findings lend support to the theory that roentgen therapy destroys one or more follicles which have matured, but which have failed to rupture and progress through a normal corpus luteum phase. It is conceivable that x-ray destruction of these mature follicles allows a new crop of follicles to develop normally thereafter in a majority of cases.

We do not suggest either quantitative or qualitative differences in the estrogens or in the progestin produced before or after treatment with x-ray. Our studies have not included bio-assays of the various hormones in blood and urine. We have chosen endometrial biopsy because we believe it to be a reliable index of progestin activity, and because the techniques of bio-assay are difficult, expensive, and frequently inaccurate.

Partly because of Hartman's work previously referred to regarding the effect of roentgen therapy in subcastrative doses to the pituitaries of anovulatory monkeys, we feel that the essential in treatment of anovulatory flowing lies in radiation of the ovaries alone. Our latter cases have been so treated (ovaries alone) with no apparent difference between their course and the course of those with both ovarian and pituitary irradiation.

In our cases, regular ovulatory cycles began within 2 to 10 weeks following the first x-ray treatment. In the majority, they began within 4 weeks. No apparent harm resulted when a subsequent series of treatments with the same dosage was given after an interval of 2 or 3 months. However, this proposed roentgen

therapy should be used cautiously in cases in which the patient is 35 or more years old. Ovarian function already faulty, was temporarily or completely destroyed by this small dosage of x-rays in 4 patients in this age group (Cases 14, 23, 26, and 27).

SUMMARY AND CONCLUSIONS

Twenty-seven cases of menstrual abnormality with and without associated sterility are presented. The nature of flow has been evaluated by endometrial biopsy in an effort to determine whether flowing has been of the ovulatory or of the anovulatory type. Subcastrative dosages of x-ray have been employed in an effort to re-establish the normal balance of ovarian function. Endometrial specimens taken before and after x-ray treatment have been used in estimating the value of x-ray treatment to produce ovulation in those patients who do not ovulate or who have only occasional ovulatory cycles. No harmful effects from this treatment upon subsequent pregnancies have been found in the literature.

Our results indicate that the use of subcastrative doses of roentgen rays is more effective than is the present endocrine treatment. Of the 22 cases of anovulatory flowing, or of infrequent ovulatory cycles, 12 or 54.5 per cent, had 3 or more ovulatory cycles following treatment. In 3 of these patients, or 13.6 per cent, pregnancy occurred within a short time following treatment.

We have found, contrary to the experience of other investigators, no change after this dosage of x-ray in 5 patients with previous normal ovulatory cycles who had the associated complaints of sterility, dysmenorrhea, profuse menstruation, or too frequent menstruation. We believe that x-ray treatment is indicated only when there is present definite ovarian dysfunction, specifically in those patients who habitually fail to ovulate, as shown by the absence of progestin effect on the endometrium.

Three doses, each of 50 to 60 r's, given over the ovaries seem harmless to the ovaries of women under 35 years of age. Women older than this may suffer temporary or permanent cessation of follicular function.

We are of the opinion that x-ray treatment of the pituitary offers no additional therapeutic benefit.

To explain the good results we postulate a destruction by x-ray of persisting mature follicles, thus allowing a new cycle of follicle development and maturation to take place.

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A ROENTGENOLOGICAL STUDY OF INTESTINAL OBSTRUCTION

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RENEWED interest in the entire subject of intestinal obstruction has been aroused since the publication of the illuminating articles and recent book by Wangensteen on this subject. He has given us a better idea of what occurs and has elucidated a new thought in the method of treatment. He emphasizes that to carry out his plan constant observation is required. In this paper we are attempting to bring out some refinements that are possible in the x-ray diagnosis of intestinal obstruction which are of considerable aid in following such cases clinically and of help in instituting proper therapy.

That an x-ray examination is of value in the diagnosis of intestinal obstruction has been known for a long time. The literature on this subject is extensive, and among the best works is that of Laurell. While well known to the roentgenologist, the aid that this procedure can give has not been fully utilized by the clinician. It should not be stated that these observations are always absolutely diagnostic but when they are considered together with the clinical symptoms we have found the x-ray findings to be of value in giving a clue as to the type of obstruction and the underlying cause. Close co-operation between the surgeon and the roentgenologist is essential so that all findings may be properly evaluated and emphasized.

The use of barium as a contrast medium to outline the gastro-intestinal tract is a well known procedure. Not as familiar to many is the fact that gas is a contrast medium and will outline the gastro-intestinal tract. It is this contrast of gas, accumulating rapidly in intestinal stasis, which gives the diagnostic criterion for the study of intestinal obstruction. With the accumulation of gas is the accumulation of fluid. Careful study is made of the relation of the gas and fluid in appearance, position, and mobility to differentiate the type and location of the obstruction. We wish to state that in this paper we are not discussing those obstructions due to strangulation of the bowel, the result of vascular occlusion. An observation not generally appreciated is that not infrequently in a strangulated obstruction we do not find distention of the intestinal loops proximal to the obstruction either on the x-ray plate or at operation. This will be discussed in a subsequent paper.

For the purpose of this paper we divide intestinal obstruction into two types: the first is that due to a block of the free passage of gas and fluid in the bowel—the mechanical or dynamic obstruction; the other type is of neurogenic origin and is due to a stimulation of the sympathetic nervous system which is inhibitory to the bowel. In such cases there is interference with the propulsion mechanism, with a lack of peristalsis and resultant failure of transportation and absorption of intestinal contents. Wangensteen calls this inhibition ileus. It is more popularly known as paralytic or adynamic ileus. Among the causes of paralytic ileus are infection, both intra-abdominal and extra-abdominal; trauma, both generalized and operative—traction on the mesentary; cord lesions; shock; severe pain such as ureteral or gall-stone colic; and psychic factors, such as fright. We can often differentiate the paralytic distention due to intra-abdominal infection from the other larger groups.

Let us again make clear that the differentiation between dynamic, mechanical obstruction, and adynamic, paralytic, ileus, and also between the two types of paralytic ileus are not always absolute. A great difficulty is that we may have combinations of the mechanical and the paralytic. If a simple mechanical block is allowed to continue, the distended

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bowel may become non-viable from the interluminary pressure. We will then have the same result as that of a strangulated bowel. On the other hand, a paralytic ileus caused by a localized peritonitis may cause a mechanical block by the production of fibrinous and organized adhesions. These points are emphasized in order to stress the necessity for frequent x-ray examinations during the course of intestinal obstruction and, as Wangensteen points out, this becomes particularly essential when operation is postponed and conservative medical treatment is instituted.

Both types—mechanical and paralytic—have one thing in common, namely, loops of bowel distended with gas and fluid. What is the origin of the gas? Except in infants, gas is normally found only in the stomach, duodenal bulb and colon. We can look to three sources for the gas in the bowel. Air is continually being swallowed. Normally, this is quickly broken up into small bubbles in the jejunum and carried in the intestinal contents. Gas also accumulates as a result of bacterial fermentation and there is an exchange of gas between the bowel and the circulating blood. Wangensteen believes that in the obstructed bowel swallowing of air accounts for 68 per cent of the gas; 22 per cent is due to diffusion from the blood stream and 10 per cent the result of digestive fermentation. Normally this gas is not visible in the small bowel on the x-ray films, as it is carried in solution. But as soon as stasis occurs either from a mechanical block or lack of propulsion, the gas is thrown out of solution and can be seen on the film. More gas accumulates, there is interference with absorption, so that a vicious circle is started. The gas produces distention; distention interferes with absorption, which leads to production of more gas. In addition to the gas, fluid accumulates as a result of diminished absorption and increased activity of the secretory glands. Rowntree estimates that 7000 cubic centimeters of fluid will normally reach the bowel each day. This fluid is made up of saliva, gastric juice, pancreatic juice, and succus entericus.

These two findings of gas and of fluid in the small bowel are diagnostic of an intestinal obstruction.

Before discussing the x-ray findings in intestinal obstruction let us first visualize the intestinal tract as a series of communicating loops between which there is a free movement of gas and fluid within the lumen. In any given position the fluid will tend to gravitate to the dependent parts and the gas will rise. It is this movement of gas and fluid as well as the character of the loops that we use as our criteria for the differential diagnosis between the various types of obstruction.

DYNAMIC OR MECHANICAL OBSTRUCTION

With the onset of a mechanical obstruction the bowel above dilates due to the accumulation of gas. The appearance of the bowel on the film will depend on its location. When the upper loops are distended the typical "herringbone" or "coiled spring" appearance will be present. This is due to the stretching of the circular folds or valvulæ conniventes. The valvulæ conniventes are reduplications of the mucous membrane, the two layers of the fold being bound together by submucous tissue. These folds are permanent and are not obliterated by distention. They run around the intestine at right angles to the long axis. When distended, the jejunum and upper ileum tend to lie in a horizontal direction one above the other and give the "stepladder" appearance. As we descend the ileum the circular folds diminish and are absent in the lower ileum. Distention of the lower ileum will have certain characteristics which will distinguish these loops from jejunum and upper ileum. Because of the absence of the valvulæ conniventes the wall will be smooth in contra-distinction to the "herringbone" appearance of the upper loops. The distended loops do not lie parallel but take a vertical direction unless prevented by adhesions. The distended smooth walled vertical loops may be confused with a distended loop of sigmoid.

A short discussion of the appearance of gas in the large bowel will not be out of order because of the difficulty sometimes present of differentiating between the large and small bowel on the x-ray film. Distention of the colon will show the characteristic haustral markings and can also be recognized by its position on the abdominal film. The ascend-

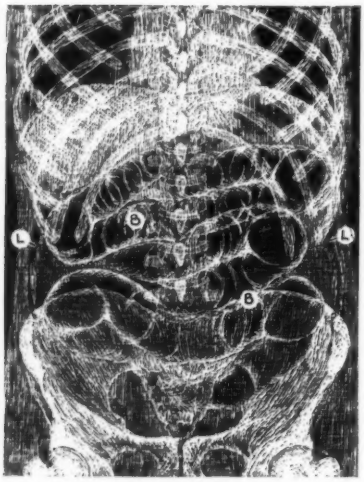

Fig. 1a. Anteroposterior view with patient supine. Dynamic—mechanical—small bowel obstruction. *B*, Distended small bowel; *L*, Properitoneal fat line. Distended loops of small bowel proximal to point of obstruction.

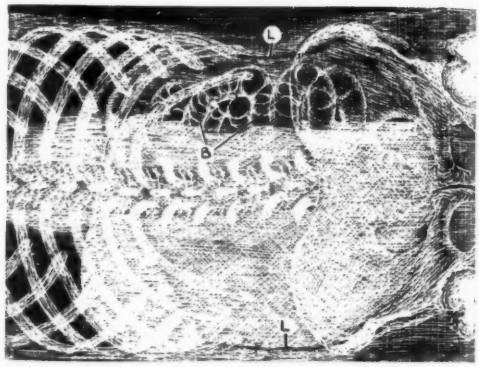

Fig. 1b. Left horizontal view. Dynamic—mechanical—small bowel obstruction. *B*, Distended small bowel; *L*, properitoneal fat line. Free movement of gas and fluid in the loops of small bowel. Fluid tends to lie in the same horizontal plane.

ing and descending colons lie vertically in the flanks. If doubt arises as to whether or not the distended bowel is colon, a barium enema can be safely given and the nature of the distended loop determined. The frequent taking of films will also help in distinguishing the nature of the bowel, as the gas often shifts and at times the bowel will appear more characteristic than at other times. In the colon a differential point can often be made between a slowly growing obstruction and a sudden obstruction. In the slowly growing obstruction, especially in the sigmoid, the back pressure will dilate the cecum which may distend to the point of perforation (Saeltzer and Rhodes). Distention in a volvulus will be limited to the twisted loop which may be dilated to such a degree as to completely fill the abdomen. The distended sigmoid rises vertically, starting in the midline. If allowed to continue, the volvulus will act as a mechanical block to the proximal colon.

The question arises as to whether an obstruction of the large bowel can distend the ileum. Wangensteen states that the ileocecal valve prevents the return of fluid or gas from the colon into the ileum and he makes a differential diagnosis between the small and large bowel obstruction by absence of vomiting in the latter. This has not always been our experience. We have encountered several cases of large bowel obstruction in the colon with an incompetent ileocecal valve, thus allowing distention of small intestine and vomiting of large quantities of fluid. It was noted in these cases that the patients did not exhibit the toxicity seen in small bowel obstruction and the distention was not as marked.

ROENTGEN FINDINGS IN MECHANICAL OBSTRUCTION OF SMALL BOWEL

With experience one can diagnose a mechanical obstruction of the small bowel from a single flat film. The loops of obstructed bowel are large and dark. The circular folds are prominent, giving an impression of a spring ready to uncoil. There is a marked contrast between the gas-distended bowel and the rest of the film. It has that dynamic quality of light and shade and is comparable to those paintings in which the artist uses light and shade with harsh contrasting lines to convey the impression of activity and restlessness. The coils have sharp hairpin turns and as a result of peristalsis some of the coils will be indistinct from movement during the exposure. The term "dynamic" is truly expressive of the appearance of this bowel. The distention involves only that part of the bowel proxi-

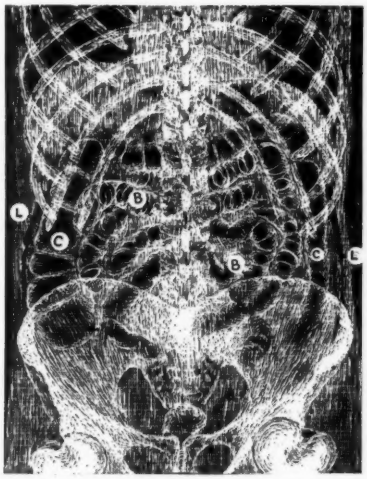

Fig. 2a. Anteroposterior view, supine. Adynamic—paralytic, inhibition—ileus. *B*, Distended small bowel; *C*, distended colon; *L*, properitoneal fat line. Distention of small and large bowel.

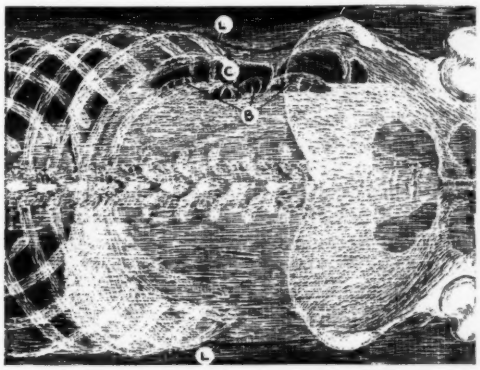

Fig. 2b. Left horizontal view. Adynamic—paralytic, inhibition—ileus. *B*, Distended loops of small bowel; *C*, distended colon; *L*, properitoneal fat line. Free movement of gas and fluid in the bowel. Fluid in the loops tends to lie in the same horizontal plane.

mal to the point of obstruction. The loops of distended bowel are large or tend to become so in future examinations.

As the condition continues, fluid accumulates within the bowel. There is a free movement of gas and fluid. The gas tends to collect in the upper loops and the fluid gravitates to the dependent position. Examination with patient in the supine position does not show the fluid present. Fluid is demonstrated only by taking films with the patient upright or lying on the side and the film taken with the ray directed horizontally. We call this latter position right or left side horizontal—R.S.H. or L.S.H. These positions allow the gas to rise above the fluid and show horizontal fluid levels. Additional lower fluid levels may be seen if gas is trapped in shorter loops, but the fluid tends to lie in a horizontal plane. (Fig. 1.)

Free fluid may be present in the peritoneal cavity. This is evidenced by increased density between the loops of bowel and the absence of the properitoneal fat line.

In an effort to overcome the obstruction, vigorous peristalsis is present. This is evidenced on the film by change in size and shape of the gas filled bowel in subsequent examinations, besides the blurred outline of some of the loops undergoing peristalsis at the time of the exposure. The peristalsis also tends to cause the gas to collect in fewer and larger loops. As the obstruction continues fluid increases in the lumen of the bowel; the bowel wall becomes edematous, and contrast of the gas against the bowel will be less sharp. As result of interluminary pressure the bowel wall may become non-viable and peristalsis cease. The mechanical dynamic obstruction will then pass into a paralytic state. Differentiation between the two at this point may be difficult.

A mechanical obstruction of small bowel may be complete or incomplete. If partial, gas may be found in different parts of the colon but never under tension. When the obstruction is complete, the large bowel is comparatively free of gas, unless enemas have been given.

The diaphragm moves freely in a mechanical obstruction and is limited only by the amount of the distention.

To recapitulate, the x-ray findings in a mechanical obstruction are large loops of gas distended bowel, accentuated peristalsis, free movement of gas and fluid with fluid levels tending to lie on the same horizontal plane. The movement of the diaphragm is limited only by the distention.

ADYNAMIC OBSTRUCTION—PARALYTIC ILEUS

Roentgen findings in paralytic ileus (adynamic ileus, reflex inhibition—Wangensteen) result of stimulation of sympathetic nervous system. As previously stated, this condition is due to a lack of propulsion of the intestinal contents. It occurs in shock, fright, ureteral colic and gall-stone colic; in short, any severe abdominal pain which may influence the autonomic nervous system. This form of distention is what we see so often after operation. It is not due to a paralysis of the muscle but is due to stimulation of the sympathetic nervous system which is inhibitory to the muscle of the bowel. Blocking the sympathetic system by spinal anesthesia relieves the distention and peristalsis is resumed.

Too often a postoperative distention is considered a paralytic ileus, which is expected to clear up in a few days. A mechanical block may be present, however, and be overlooked. The difficulty of making this differential diagnosis is one of the causes of the high mortality in obstruction. It is for this type we especially make a plea for early and frequent x-ray examinations. It is possible by this means to differentiate the two conditions with a fair degree of accuracy.

What diagnostic criteria have we in the x-ray examination of paralytic ileus other than that due to peritonitis? First, we have distention. Unlike the mechanical obstruction, in which distention is above the point of obstruction, in paralytic ileus we have a segmental distention with involvement of the small and large bowel. The film in the supine position shows distention of bowel without the sharp contrasting lines. It is flat and the picture is one of rest. The condition is appropriately called adynamic. The distention is segmental. We find loops of small and loops of large bowel scattered over the abdomen. The distention may vary from the normal sized bowel to a very marked dilatation. The presence of fluid is variable. There is free movement of gas and fluid with change in position of patient. A most important clinical sign is the absence of peristalsis. While this is not a roentgenological diagnosis, it is noted that subsequent examinations within the hour show little change in the relative positions of the distended loops, in this manner indicating the lack of activity of the bowel.

Paralytic ileus as a result of localized and generalized peritonitis. The appearance of this type of paralytic ileus on the x-ray film has certain characteristics which distinguish it from the paralytic ileus without peritonitis. At the beginning when the peritonitis is localized the appearance on the film may be one of a few dilated loops of bowel of an adynamic quality adjacent to the site of the peritonitis or localized abscess. If the peritonitis spreads and becomes generalized more bowel becomes distended, and the x-ray picture changes. Distended loops of bowel with gas and fluid are present and we have the picture of an adynamic ileus. In addition, with peritonitis, we may have accumulation of fluid in the abdomen. This is indicated on the film by the increase in the shadow noted between the loops of bowel.

Another more important sign may be present. This is the appearance of the properitoneal fat line. The peritoneal lining consists of a thin wall of endothelial cells. Next to this layer is a layer of fat containing some connective tissue. This fat lies between the peritoneum and muscle. It varies in thickness in different individuals. It can be seen on the film as a dark stripe in the flanks next to the bowel. With accumulation of fluid in the abdomen or edema of the peritoneal endothelial cells there is an edema of this fat layer. This brings the absorptive capacity of roentgen-ray of the fat layer up to the neighboring muscle and the contrasting dark stripe is then lost. This results in a homogeneous shadow in the flank.

Care must be taken in evaluating the presence of this fat line. Its presence does not exclude peritonitis. Its absence requires study in order to be sure that the film was not overexposed. When absent it indicates fluid in the abdomen, which may not necessarily be fluid from a peritonitis. As previously mentioned, fluid may be present with a mechanical obstruction and the line be lost. It is of greatest diagnostic significance when absent in only one part of the flank. If associated with a local paralytic ileus it is diagnostic of a localized abscess or localized peritonitis.

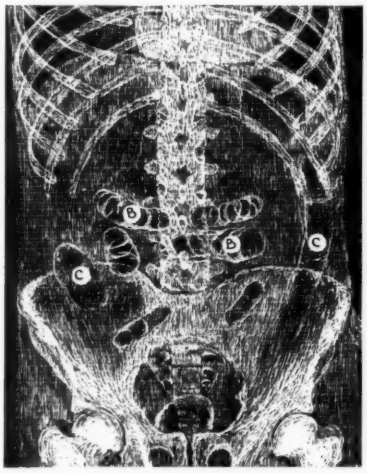

Fig. 3a. Anteroposterior view, supine. Adynamic—paralytic—ileus as the result of generalized peritonitis. *B*, Distended loops of small bowel; *C*, distended loops of colon. Scattered loops of distended small and large bowel; absent properitoneal fat line.

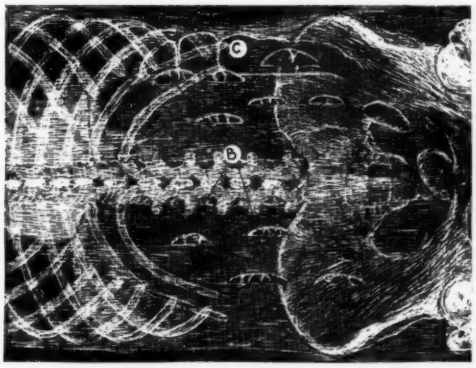

Fig. 3b. Left horizontal view. Adynamic ileus as result of generalized peritonitis. *B*, Distended loops of small bowel; *C*, distended colon. Distended loops of small bowel scattered over abdomen, fixed in position resulting in multiple fluid levels. Absent properitoneal fat line.

Localized peritonitis. The paralytic ileus present in a localized peritonitis or abscess may consist of a single loop or may involve a few loops. We have not infrequently made a diagnosis of a walled off abscess by the presence of a few loops of adynamic distended bowel neighboring a region with an absent properitoneal fat line.

Generalized peritonitis. The appearance of the gas filled bowel on the x-ray film in generalized peritonitis also differs from that of mechanical obstruction and non-peritonitic paralytic ileus. The loops of bowel as in the non-peritonitic paralytic ileus are distended with gas and contain fluid levels. They are segmental in nature and loops of large bowel and loops of small bowel are equally distended. That is, the distention does not extend only to the point of obstruction but is scattered. Peristalsis is absent or diminished so we do not have the tendency of the small loops to gather into fewer larger loops; consequently, the loops are small and shallow. If the peritoneal exudate is sticky or plastic, the loops are relatively fixed in position. Turning the patient upright and to the left and right horizontal position (L.S.H. and R.S.H.) demonstrates the fixed position of the loops of bowel. There is no free movement of gas and fluid; consequently we have a scattering of loops with multiple fluid levels. Peristalsis is absent, as it is in all paralytic ileus.

There is a limitation of movement of the diaphragm which may be limited only to the side of the peritonitis. (Fig. 3.)

To summarize, in a local peritonitis or walled off abscess there may be an ileus consisting of a single or multiple loops of bowel at the site of the infection, and obliteration of the properitoneal fat line. In generalized peritonitis we have a distention of the bowel, which may involve the large as well as small bowel, of an adynamic nature, with loops scattered over the abdomen. Peristalsis is absent; there is limitation of movement of the diaphragm on the affected side or on both sides if generalized. The loops of distended bowel are small, shallow, and relatively fixed in position. This is seen on the x-ray film by multiple fluid levels scattered over the abdomen. There is no tendency toward a few large loops. There is fluid between the loops and likewise there may be an obliteration of the properitoneal fat line.

A word of caution should be said in interpreting the course of a case of ileus. A stomach tube may be passed and suction applied. The distention may be less and the patient

apparently be improved. A false sense of security is held by the clinician and the patient. It is at this point that follow up x-rays are most important. The release of distention modifies the symptoms by draining the stomach and some of the fluid contents of the small bowel. The obstruction, however, may be unrelieved and if allowed to go on may result in a strangulated bowel. X-ray examination may show the mechanical block to be still present.

The use of the Miller-Abbott tube is an additional aid both in diagnosis and treatment. In our own hands, when passage is possible within a reasonable time, it has cleared up a difficult diagnosis and avoided the necessity of operation. Certain inherent dangers connected with the use of the tube which should be emphasized:

1. The difficulty in passage causes a loss of time and the delay in operation may be vital.

2. The use of the tube may relieve the symptoms without causing release of the obstruction. If a simple obstruction is present, no danger results but if there is a strangulation obstruction or if the simple obstruction finally interferes with the blood supply during this period of observation, vital time is lost in operative delay.

3. We have had an interesting and unusual experience in one of our own cases which is worthy of note. The tube relieved the distention and symptoms for a time but in its descent it carried some of the collapsed proximal bowel into the distended bowel and caused a small bowel intussusception.

TECHNIQUE OF THE EXAMINATION

The technique of taking the films does not involve any added discomfort to the patient. If the patient cannot be brought to the x-ray department, satisfactory diagnostic films can be obtained at the bedside.

The first film is taken with patient in the supine position. These patients are uncomfortable to lie on the distended abdomen for a film in the prone position. This film gives general information as to the distention and may be sufficient to establish the diagnosis. This position does not show fluid levels. If possible, a film should be taken with the patient upright.

Another film is taken with the patient lying horizontally on either side. This will show fluid levels and movement of gas and fluid. If the patient is too ill to be placed in the erect position, the same information regarding free movement of gas and fluid can be obtained by the horizontal exposures with the patient lying first on the right side, and then on left side.

We have introduced the following terms and their abbreviations to designate the position of the patient and the directions for ordering special examinations for cases of bowel obstruction. The usual request for film of the abdomen in the prone, supine, or upright position is standard terminology and of common usage. There is no commonly accepted term used to direct films to be taken in the anteroposterior or postero-anterior projection with the patient lying on his side. To facilitate the transmission of orders to the x-ray department we suggest the term to be employed for such a view as "side horizontal." This can be abbreviated "S.H." As we are interested in the side uppermost rather than the side on which they lie, we can add "R." for right, which implies right side up; and "L." for left.

Directions can thus be expedited and orders issued as take films of abdomen in "A.P. and L.S.H. (or R.S.H.)" views.

The usual film overexposes the flanks. To study the properitoneal fat line it is necessary to have soft tissue detail. Films are underexposed, or a medium, as an aluminum wedge, is inserted in the space between the flank and the film to bring up the density of this part to that of the abdomen.

Just as the surgeon may not make the diagnosis of an obstruction with one examination, but may examine repeatedly, so may it be necessary to have repeated x-ray examinations. Obviously it is erroneous to correlate the x-ray findings with a physical examination done a few hours or a day later. Just as the clinical picture changes from hour to hour so will the x-ray findings change and the clinical findings must be correlated with the x-ray findings of that time.

CONCLUSION

Properly performed x-ray examination correlated with a careful history and physical

examination can be of great aid in confirming a diagnosis of intestinal obstruction. There is certain evidence found in the x-ray examination of cases of mechanical obstruction, paralytic ileus of reflex inhibition, and paralytic ileus the result of peritonitis, which if present and properly evaluated will differentiate these three conditions.

Let us make clear that the observations on intestinal obstruction which we have made in this paper are not always present or always absolutely diagnostic. But, taken together with the clinical symptoms and frequent observations on the patient, we have found them to be of great value in giving both a clue as to the type, the location, and at times even an opinion as to the underlying cause of the obstruction. We have had cases of mechanical obstruction with gangrenous bowel and negative findings on the x-ray. We have also had cases of a generalized peritonitis in which no distended loops of bowel were seen on the film. We have had cases in which fluid was present and the properitoneal line was unchanged. We do not know why some cases have these negative findings; but when the above positive findings of gas and fluid are present, careful study of the films correlating the x-ray findings with the symptoms at the time and repeating the examination when necessary, will make it possible often to arrive at the correct diagnosis and to institute the proper therapeutic procedure.

We wish to express our thanks to Dr. Leon Goldman of the surgical department, University of California, for his criticism and suggestions in preparation of this paper and to Jean Kelso, the artist who prepared the illustrations.

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A COMPARISON OF THE FIBRINOGEN AND PROTHROMBIN LEVELS OF MATERNAL AND CORD BLOOD AT DELIVERY

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DURING a study of the so called prothrombin time of maternal and cord blood plasma (16), the fibrinogen content of each was determined simultaneously in a number of cases. The findings furnish the basis of this report. Isolated studies on fibrinogen levels under various conditions are not infrequent in the literature. Unfortunately, these results are seldom comparable because of the widely differing techniques employed.

Among the earliest reports on the fibrinogen content of fetal blood is that of Krueger. The fibrinogen values of whipped blood from the fetus at the time of birth were found to be considerably lower than in the mother. The mothers gave values slightly higher than normal. Landsberg, in a more extensive study, found a similar relationship between the fibrinogen values of the non-pregnant, the pregnant, and the cord blood. In 1922 Gram (8) presented the results of a new gravimetric method based on the precipitation of fibrin from physiological salt solution. During pregnancy he noted a rise in plasma fibrin well above the average which reached a peak shortly after delivery. Foster (6) in the same year confirmed Gram's work. Foster and Whipple (7), in a study of the influence of diet on blood fibrin levels, gave the normal range of plasma fibrin as 0.25 grams per cent to 0.50 grams per cent. Chandler, using a micro-Kjeldahl method in place of gravimetric measurement, reported on 39 cases including pregnant and non-pregnant women. The former gave high fibrin values. Kvater and his colleagues, using a salting out method, reported in 1926 an increase of plasma fibrinogen during pregnancy followed by a sharp drop at the time of delivery to almost one-half the normal value. The newborn fibrinogen levels were reported as low. Achard and his colleagues described a low total plasma protein in cord blood and suggested that this was due to a deficient globulin fraction. Naeslund, employing a refractometer, found the amount of plasma fibrinogen to be low in healthy children at birth. A subsequent sharp rise was exhibited during the first few weeks of life. Crane and Sanford, in 1936, described the variations in fibrinogen content of the normal infant. They made 131 determinations on 10 infants over a period of 10 days. In a majority of cases the plasma fibrin content lay between 0.22 grams per cent and 0.55 grams per cent. The average for the first day was 0.29 grams per cent. They described a rise during the first 4 days of life that levelled off with no consistent change thereafter. The results of other workers generally confirm the finding of low normal fibrinogen levels in the newborn and higher than normal in pregnant women at term.

Recently a few reports on the prothrombin levels in the newborn have appeared. The results of Brinkhous, Quick (19, 20), Waddell (22, 23), Owen, and our own findings, with but few exceptions, indicate that low values for cord prothrombin are the rule. Hellman and Shettles, in a recent publication, suggested that prematurity is also associated with low prothrombin levels.

It was the purpose of this study to determine whether the fibrinogen values of maternal and cord blood were altered sufficiently to affect the prothrombin clotting times. A total of 18 deliveries at the Philadelphia Lying-In Hospital were studied. Six healthy adult males were used for control observations.

METHODS

At the time of severing the cord, blood from the placental stump was collected directly

From the Ayer Clinical Laboratory of the Pennsylvania Hospital, Philadelphia, Pennsylvania.

into a paraffin lined beaker. Immediately a small test tube containing 0.5 cubic centimeter of 0.1 molar sodium oxalate solution (approximately 1 per cent) was filled to a 5 cubic centimeter mark with blood from the beaker, and the contents gently mixed. Shortly thereafter a similar sample was collected from an antecubital vein of the mother by means of a clean, dry syringe. The prothrombin content of each was determined after the method of Quick (18). The plasma was obtained by centrifuging for 20 minutes or more at approximately 3,000 revolutions per minute. The volume of packed red blood cells was determined simultaneously. One cubic centimeter samples of plasma were taken for fibrinogen determinations. The technique[1] was a modification of that presented by Gram (9). It consisted of diluting 1 cubic centimeter of plasma with 9 to 19 volumes of physiological salt solution in a large test tube and adding 0.1 cubic centimeter of calcium chloride solution (0.025 molar). The contents were then mixed and the tube was permitted to stand in a water bath at 37 degrees C. for at least 2 hours. In most instances a watery, gelatinous clot formed which easily could be transferred in its entirety from the test tube to several layers of filter paper. The fibrin clot was then washed with physiological salt solution to remove nitrogenous contaminants. The nitrogen content was determined by the micro-Kjeldahl method as described by Koch and McMeeken. The protein content was estimated by multiplying the nitrogen figure by the factor 6.25. To determine the percentage fibrinogen of undiluted plasma a correction had to be made according to the formula of Gram (9). Observed readings for volume of packed erythrocytes were used in calculating maternal and cord blood while 46 volumes per cent was arbitrarily selected for correcting the normal controls.

RESULTS

Maternal blood plasma furnished large, firm, cohesive clots. The fibrinogen content in grams per cent ranged from 0.25 to 0.66 with an average of 0.54. The prothrombin content averaged 130 per cent of the normal controls.

[1]Indebtedness is expressed to Dr. Leandro M. Tocantins for suggesting this method.

Cord plasma as a rule furnished delicate, loose, friable, watery clots. In 2 instances no clots at all were perceptible. This occurrence was found to be associated with an excessive dilution with saline (19 volumes) rather than a deficiency of fibrinogen or prothrombin. The fibrinogen values varied between 0.23 and 0.49 grams per cent with a mean of 0.33. The prothrombin content averaged 68 per cent of the normal controls.

Adult male plasma formed clots that resembled those of maternal plasma yet failed to duplicate the dense tough character of the latter. The fibrinogen content varied from 0.22 to 0.33 grams per cent with a mean of 0.275. As this plasma was used as normal prothrombin control material, the prothrombin content was taken to be 100 per cent.

EVALUATION

Eagle and Quick (21) describe the effect of varying concentrations of fibrinogen on the clotting time. They conclude that, except in extremely low ranges of fibrinogen, the clotting time of plasma is unaffected. This conclusion is borne out by our findings. Comparing the fibrinogen and prothrombin values of infants with normal adult males, one sees that with nearly identical fibrinogen levels there are widely differing prothrombin clotting times; the infants are significantly below normal in the latter respect. On the other hand, on comparing the plasma from mother's blood with that of normal males, one sees an increase in the fibrinogen content associated with an acceleration of the prothrombin clotting time above normal. With these findings in view, it becomes difficult to attribute to the fibrinogen content of the plasma any significant influence on the prothrombin value. However, it is interesting to speculate whether the frequent association of low prothrombin in the presence of normal fibrinogen levels represents a developmental weakness in liver function or a vitamin deficiency.

SUMMARY

1. The plasma fibrinogen and prothrombin levels of 18 samples of maternal and cord blood taken at the time of delivery are compared to the values of 6 normal individuals.

2. The fibrinogen content of plasma of cord blood is normal but the prothrombin content is reduced.

3. Fibrinogen and prothrombin contents of maternal blood at the time of delivery are above normal.

4. The prolonged prothrombin clotting time of cord blood is not due to a deficiency of the plasma fibrinogen.

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EFFECTS OF EXPERIMENTAL CHRONIC HYPERPARATHYROIDISM ON THE KIDNEY OF THE DOG

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AFTER the discovery of the parathyroid glands by Ivor Sandstrom in 1880, no attention was paid to them until their rediscovery by Gley in 1891. Moussu, in 1897, and later Halsted demonstrated the functional independence of the parathyroid gland and the thyroid. Because extracts of the gland were not available at that time, an erroneous hypothesis was developed as to the action of the parathyroid glands. Although various types of bone lesions were described by earlier investigators, the first anatomical descriptions of such lesions in parathyroid overfunction were ascribed to Stansky by Lievre. It was Askanazy and Lundborg, in 1904, and Chvostek, in 1908, who paved the way for an early understanding of the parathyroid glands and their relationship to bone lesions.

It was not until 1905, however, that MacCallum associated the parathyroid glands with renal disease, and showed the coexistence of tumors of the parathyroids with pathological changes in the kidney. Barker mentions a case studied by Thomas and Wentworth in which parathyroid hyperplasia was associated with chronic renal disease of 10 years' standing. In this patient deposits of calcium were present in other parts of the body, and the x-ray showed calcification of smaller arterioles. At autopsy bone changes were present. Bergstrand described hyperplastic enlargement of the parathyroid glands in 10 cases of severe renal disease. None of these patients showed osteoporosis or osteomalacia but did show deposits of calcium salts in the kidney.

The entire subject of hyperparathyroidism has now been thoroughly investigated by numerous workers, both clinically and experimentally. The work of Mandl, in 1926, and of Hannon, Shorr, McClellan and DuBois, in 1930, showed the relationship between overfunction of the parathyroid gland and osteitis fibrosa cystica, and in 1934 the work of Albright, Aub and Bauer definitely pointed out that there were other clinical forms of hyperparathyroidism which presented symptoms in common with other disease entities often giving no signs related to bone disturbances; namely renal stones. In their series of 17 cases of hyperparathyroidism proved by operation and removal of parathyroid tumors there were 7 patients who had only symptoms of stone, 1 with only symptoms of a nephritis, and 10 who had symptoms referable to the genitourinary tract. Routine calcium phosphorous studies in all patients with stone revealed 8 to have parathyroid tumors. In a later report, in 1935, Albright and Bloomberg (2) reported a series of 23 cases of hyperparathyroidism. Eleven patients with renal stone were proved at operation to have parathyroid tumors. These patients had no bone lesions or symptoms. Only 5 of 23 patients had bone lesions or symptoms without renal disease.

Castleman and Mallory, in 1935, reported that, in patients with hyperparathyroidism associated with stone, symptoms had lasted for an average of 3.2 years and when bone lesions were present the symptoms had lasted for an average of 8.6 years. Elsom, Wood, and Ravdin, in 1936, reported a case of hyperparathyroidism with renal insufficiency. Their case presented signs of osteitis fibrosa cystica with diffuse calcifications of the kidneys. A tumor of the parathyroid gland was found at operation. The disease in this patient escaped detection for some time because it was thought that her symptoms arose from an atypical form of glomerulonephritis.

It is interesting, however, to note the various theories for the formation of stone in the kidney. Randall in his extensive investigation as to the origin of primary renal calculi, suggests that one of the causes for such stones might be attributed to parathyroid overfunc-

From the Harrison Department of Surgical Research and the Department of Urology, School of Medicine, University of Pennsylvania.

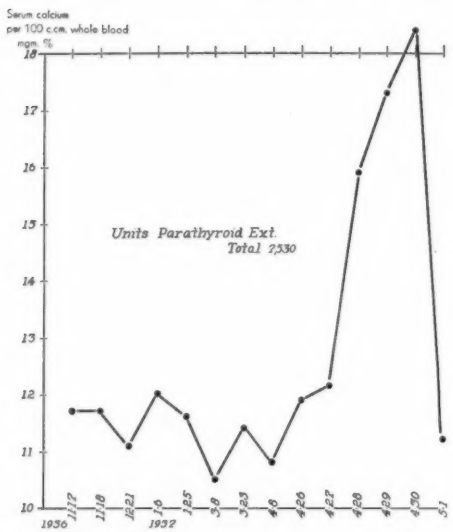

Fig. 1. Dog 97.

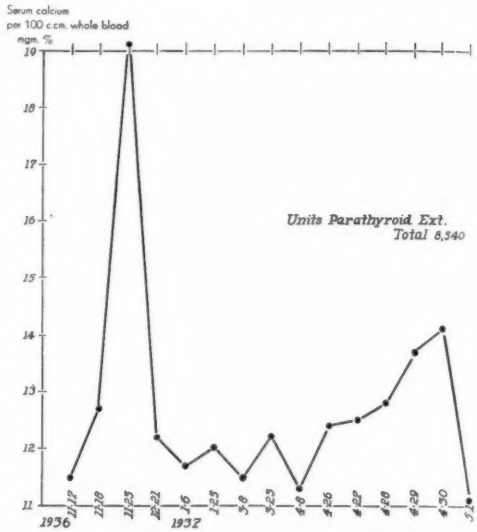

Fig. 2. Dog 103.

tion. The author has attempted to produce experimentally a condition of hyperparathyroidism in normal adult dogs by means of injecting them subcutaneously with parathormone.[1]

It was considered advisable to conduct a long continued experiment in which conditions might resemble more closely those to be expected in clinical cases of hyperfunction of the parathyroid gland.

[1]Kindly supplied by Eli Lilly & Co.

METHODS OF STUDY

Adult dogs were used in these experiments because of the probability that older dogs would show similarities in changes produced by an overfunction of the parathyroid glands as seen in human clinical cases (14). The exact ages of the dogs were not known because the animals were obtained from a dog farm where they were collected from several sources. The diet was well balanced and simulated one which any human might follow. Water was

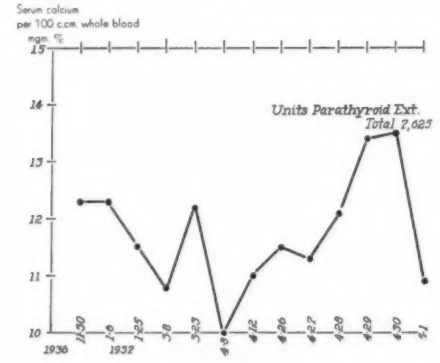

Fig. 3. Dog 125.

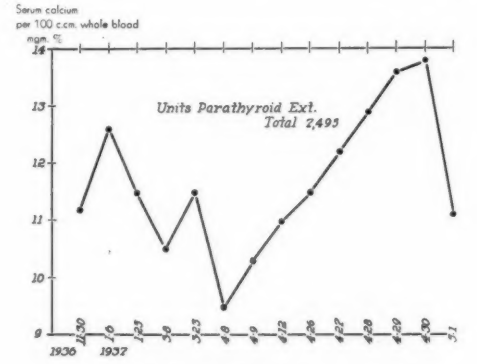

Fig. 4. Dog 131.

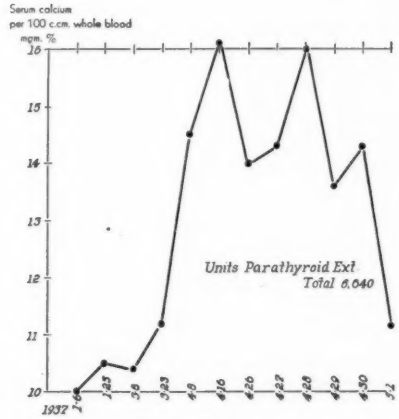

Fig. 5. Dog 215.

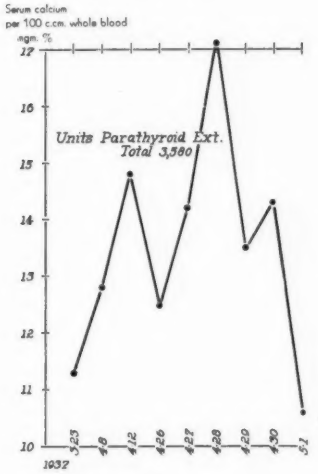

Fig. 6. Dog 523.

kept in the cages at all times. Two groups of 15 and 16 dogs, respectively, were used, one being a control upon the other. The experiments were carried on in two parts. The first was started on November 17, 1936, and ended May 1, 1937, a total of 6 months, and the second experiment from September 23, 1937, to May 4, 1938, a total of 8 months. Parathormone was used throughout the entire research. The animals received varying doses of parathormone injected subcutaneously. The dose depended on the tolerance of the dog to the hormone. At first the dose was 100 units daily, which was calculated according to the weight of the animal. However, the dose was too large, and as a result many of the animals became sick and several of them died. It was

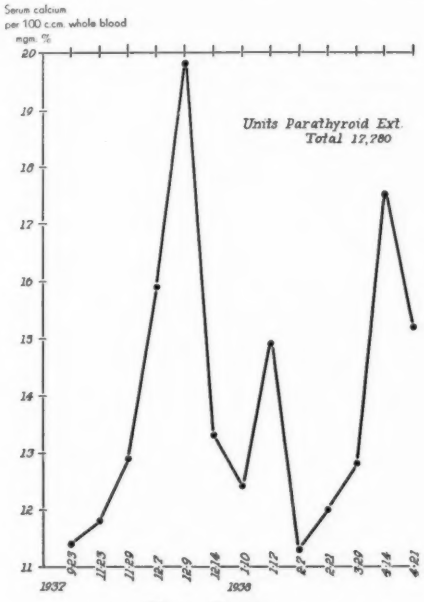

Fig. 7. Dog 43.

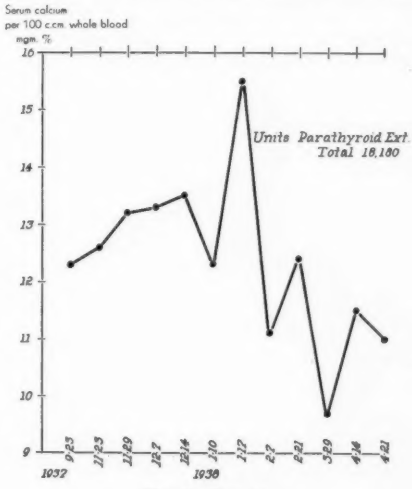

Fig. 8. Dog 45.

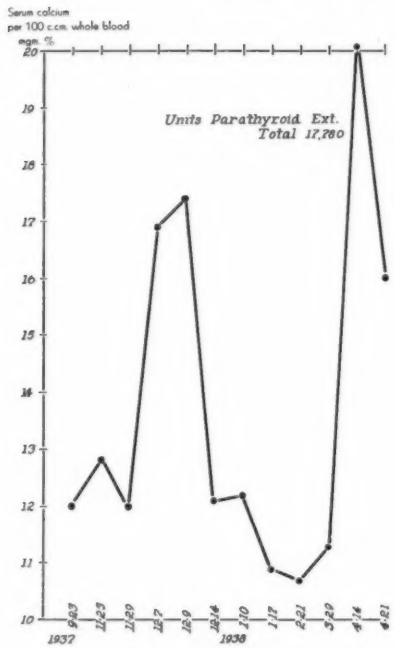

Fig. 9. Dog 47.

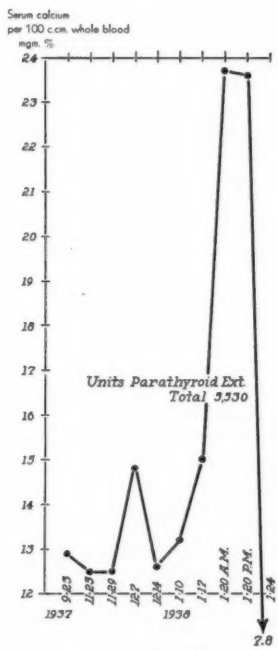

Fig. 10. Dog 68.

therefore necessary to employ the highest possible dose that would not make them sick or die. The best dose was found to be 5 units daily, increasing it to 20 units after several days, and then gradually increasing the dose at frequent intervals so that by the time the animals were ready to be sacrificed they were receiving a dosage of as high as 600 units of parathormone daily. Rest periods from the hormone were given whenever it was thought necessary. Serum calcium estimations were taken at frequent intervals, although at times this was difficult because the blood coagulated so rapidly. Viosterol[1] was added to the diet of several of the dogs, as suggested by Johnson. Other mineral studies were not attempted since much work has been done experimentally by other investigators (5, 6, 15, 23).

In several instances roentgenograms were obtained during the course of the experiment to note any renal changes. In the first group of dogs nephrectomies were done to note whether the effect of parathormone was greater on the remaining kidney.

When the animals died or were sacrificed, the organs were carefully examined grossly and microscopically. The urogenital organs were examined minutely for calcifications, especially the papilla of the kidney, which was examined for calcium plaques, described by Randall (30). The tissues removed for section were imbedded and blocked in paraffin. The stains used were the VonKossa, alizarin, Hansen Bach, and the modified Schultze technique for clearing. The whole kidney of dogs 148, 163, 43, 45, and 47, was cleared by the modified Schultze method and stained with alizarin, which is a specific for calcium (this technique is the one followed by the Wistar Institute of Anatomy in preparing skeletons of rats), and the opposite kidney was sectioned.

The parathyroids in some of the animals were fixed for section as were specimens of bone taken from the forelegs of the animals,

[1]Supplied by Mead Johnson & Co.

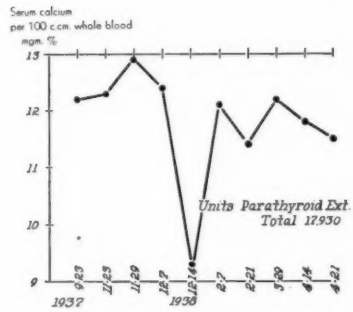

Fig. 11. Dog 163.

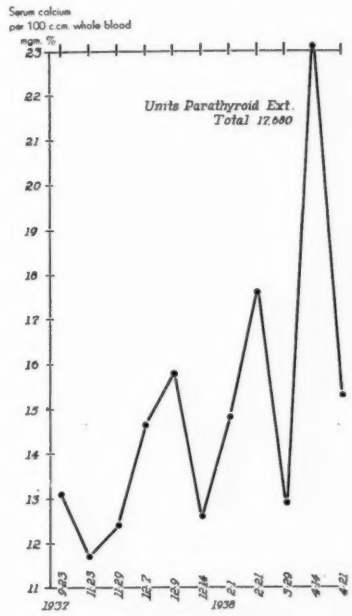

Fig. 12. Dog 148.

Chemical analysis of bladder gravel found in dogs 131, 103, and 125 was made.

Two groups of a total of 31 adult dogs were injected subcutaneously with parathormone during the course of the experiment. The animals to whose diet viosterol was added showed no demonstrable differences in their reactions to hyperparathyroidism produced by parathormone than did the animals without viosterol. Tables I and II give a résumé of the results observed. The abnormalities noted as a result of parathyroid poisoning of several of the animals which died are strikingly similar to the description of other investigators.

These symptoms usually appeared soon after injections were started, and no matter how heroic the treatment to save them, they succumbed. The animals that survived during the course of the experiment in spite of a high serum calcium did not present marked symptoms, with the exception of dog 68 which developed a hypocalcemia after a rest period of several days. The serum calcium rose to 23.7 milligrams per cent and dropped to 7.8 milligrams per cent. The other dogs developed hypocalcemia but not to such a degree. The reason for this fall will be discussed later. Figures 1 to 12 give a graphic picture of serum calcium readings. It can be observed how markedly the serum calcium rose in dogs 97, 103, 215, and 523 in the first group and in dogs 43, 44, 47, 68, and 148 in the second group. It was of interest to observe also the time difference in the rise, especially between dogs 97 and 103, although both received similar amounts of parathormone. Also, dog 163, which had received a total of 17,930 units of parathormone, had a near-normal serum calcium and even dropped to 9.3 milligrams per cent from a high of 12.9 milligrams per cent. Its normal serum calcium was 12.2 milligrams per cent. The microscopic section of the kidney of this animal showed few calcium deposits as compared with those of the other dogs, and the opposite kidney, when cleared by the modified Schultze method, did not take the alizarin stain.

It was interesting to observe that the average normal serum calcium of the animals was 11.0 milligrams per cent. The normal serum calcium varied from 9.8 milligrams per cent to 13.1 milligrams per cent.

The serum calcium of the animals treated with parathormone varied from 17.1 milligrams per cent in dog 523, which received a total of 3,580 units of parathormone and 54 cubic centimeters of viosterol, to 23.7 milligrams per cent in dog 68, which received a total of 5,530 units of parathormone and 101 cubic centimeters of viosterol. It is difficult to explain why some of the animals which re-

TABLE I.—SERUM CALCIUM DETERMINATIONS

Dog No.		97	98	99	100	101	102	103†	104	125†	131†	132	140	141	215	251	523
Started		11-17-36	11-17-36	11-17-36	11-17-36	11-17-36	11-17-36	11-17-36	11-17-36	11-30-36	11-30-36	12-4-36	12-4-36	12-4-36	1-6-37	1-14-37	3-22-37
Weight, kilos		13.8	10.7	17.0	14.0	12.0	13.8	11.0	12.8	17.8	16.9	12.2	12.6	21.6	15.0	11.8	15.9
Normal serum calcium, mgm. %		11.71	11.28	11.21	11.09	10.96	12.39	11.53	13.07	12.33	11.22	11.4	11.7	11.96	9.98	10.05	11.3
Total units parathyroid extract given		7530	600	2475	800	800	800	8540	900	7625	7495	225	6855	305	6640	800	3580
Total drops viosterol given		1345						1355		1595	1475		1045		1520		815
Serum calcium mgm. %	11-17-36	11.71	11.28	11.21	11.09	10.96	12.39	11.53	13.07								
	11-18-36	11.71	11.47	11.48	12.08	11.71	10.85	12.7	13.07								
	11-23-36				14.55	16.25		19.11	17.63								
	11-30-36									12.33	11.22						
	12-7-36			11.96								11.4	11.7	11.96			
	12-21-36	11.09		10.73				12.23						10.85			
	1-6-37	11.96		12.27				11.71		12.33	12.57		12.08		9.98		
	1-25-37	11.6		11.2				12.0		11.5	11.5		10.7		10.5	10.5	
	3-8-37	10.5						11.5		10.8	10.5		12.3		10.4		
	3-23-37	11.4						12.2		12.2	11.5		11.2		11.2		11.3
	4-8-37	10.8						11.25		10.0	9.45		11.2		14.5		12.8
	4-9-37										10.25		12.3				
	4-12-37									11.0	11.0						14.8
	4-16-37														16.1		
	4-26-37	11.90						12.4		11.5	11.5				14.0		12.45
	4-27-37	12.15						12.5		11.25	12.2				14.3		14.2
	4-28-37	15.9						12.8		12.1	12.9				16.0		17.1
	4-29-37	17.3						13.7		13.4	13.6				13.6		13.5
	4-30-37	18.4						14.1		13.5	13.8				14.3		14.3
	5-1-37	11.2						11.1		10.9	11.1				11.15		10.6
	Lowest	10.5	11.28	10.73	11.09	10.96	10.85	11.1	13.07	10.0	9.45	11.4	10.7	10.85	9.98	10.5	10.6
	Highest	18.4	11.47	12.27	14.55	16.25	12.39	19.11	17.63	13.5	13.8	11.4	12.3	11.96	16.1	10.5	17.1
Microscopic	Intratubular calcium	++++		+++				++	++	+	+			+	++		+++
	Plaques	+															
End-result		*	Died	Died	Died	Died	Died	*	Died	*	*	Died	Died	Died	*	Died	*

*Sacrificed.
†Bladder gravel.

TABLE II.—SERUM CALCIUM DETERMINATIONS

TABLE II.—SERUM CALCIUM DETERMINATIONS

Dog No.		43†	44	45‡	46	47¶	67	68	69	163§	148‖	236	338	294	295	296
Started		9–23–37	9–23–37	9–23–37	9–23–37	9–23–37	9–23–37	9–23–37	9–23–37	9–23–37	9–23–37	11–29–37	1–17–38	1–17–38	1–17–38	1–17–38
Weight, kilos		15.0	15.3	15.8	13.5	12.0	15.6	15.0	13.0	16.0	17.0		.	14.1	14.0	15.0
Normal serum calcium, mgm. %		11.4	11.1	12.3	12.0	12.0	11.8	12.9	12.3	12.2	13.1	11.8	10.5	12.3	11.0	10.7
Total units parathyroid extract given		17780	930	18180	1340	17780		5530	5830	17930	17680	1210	1000	650	1700	
Total drops viosterol given		3340	540	4710	650	4710		1620	2040	4710	4590	780	300	240	600	
Serum calcium mgm. %	9–23–37	11.4	11.1	12.3	12.0	12.0		12.9	12.3	12.2	13.1					
	11–23–37	11.8	11.8	12.6	12.0	12.8		12.5	12.1	12.3	11.7					
	11–29–37	12.9	13.0	13.2	12.0	12.0		12.5	13.0	12.9	12.4	11.8				
	12– 7–37	15.9	18.4	13.3	13.9	16.9		14.8	11.8	12.4	14.7	14.6				
	12– 9–37	19.8	18.3			17.4					15.8					
	12–14–37	13.3		13.4		12.1		12.6	11.4	9.3	12.6					
	1–10–38	12.4		12.3		12.2		13.2								
	1–17–38	14.9		15.5		10.9		15.0						12.3	11.0	
	1–20–38						23.7	23.6								
	1–24–38							7.8								
	2– 1–38												10.5			
	2– 7–38	11.3		11.1						12.1	14.8					
	2–21–38	12.0		12.4		10.7				11.4	17.6					
	3–29–38	12.8		9.7		11.3				12.2	12.9					
	4–14–38	17.5		11.5		20.1				11.8	23.1					
	4–21–38	15.2		11.0		16.0				11.4	15.3					
	Lowest	11.3	11.1	9.7	12.0	10.7		7.8	11.4	9.3	11.7	11.8				
	Highest	19.8	18.4	15.5	13.9	20.1		23.7	13.0	12.9	23.1	14.6				
Microscopic	Intratubular calcium	+	++++	++	++	++		+	+	++	++	++++		+++	+++	
	Plaques													+		
End-result		*	Died	*	Died	*	Died	Died	Died	*	*	Died	Died	Died	Died	Died

*Sacrificed.
†43 Cleared specimen +alizarin. Kidney shows diffuse calcium. Took alizarin dye.
‡45 Did not take alizarin dye.
¶47 Took alizarin dye. Kidney shows diffuse calcium.
§163 Did not take alizarin dye.
‖148 Kidney shows diffuse calcium. Heavily stained with alizarin dye.

Fig. 13. Bladder of dog, showing collection of calculi.

ceived almost three times as much parathormone did not have such an increase in their serum calcium unless they may have developed a tolerance (29). Other explanations offered are the age of the animal, an increased rate of excretion of calcium, or the hypercalcemia was counteracted by feeding (22, 35); as, for example, in dog 45 with a total of 18,180 units of parathormone and in dog 163 with a total of 17,930 units of parathormone, the serum calcium rose only to 15.5 milligrams per cent and 12.9 milligrams per cent, respectively.

A point of interest was the drop in serum calcium of all dogs, after rest periods, below the level of their normal serum calcium. Other investigators have reported similar observations (14), and interpreted the drop as being due to a reduction in the calcium reserve of the tissues, especially the bone. When parathormone injections are discontinued, the calcium is reabsorbed into the bone at such a rate that hypocalcemia results. This has been proved histologically and clinically (21, 29).

The dogs in which nephrectomies were performed showed no demonstrable change in the remaining kidney, as compared with the dogs in which no nephrectomies were done.

Roentgenographic studies on several of the animals, including intravenous urograms, were negative for bone lesions or nephrolithiasis. Greenwald and Gross (14) reported similar observations.

PATHOLOGY

Of greatest interest to the author were the findings at autopsy, especially the urogenital findings. Dogs 103, 125, and 131 had stones in the bladder (Fig. 13), the chemical analysis

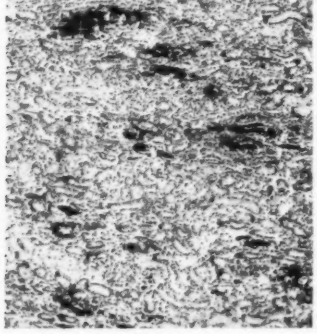

Fig. 14

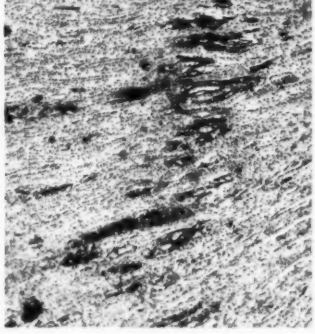

Fig. 15

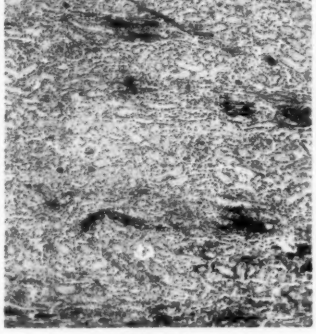

Fig. 16

Fig. 14. Photomicrograph of section of kidney of dog. There can be seen cloudy swelling and tubular degeneration. Intratubular deposits of calcium are also present. ×45.

Fig. 15. Photomicrograph of dog's kidney, showing deposits of intratubular calcium and tubular atrophy. ×45.

Fig. 16. Photomicrograph of kidney of dog, showing cloudy swelling, glomerular degeneration, and deposits of intratubular calcium. ×45.

of which proved them to be composed of calcium phosphate. The following observations, except for insignificant differences, are applicable to all the animals subjected to autopsy. There was marked hyperemia throughout the omentum and abdominal viscera. The kidneys were markedly congested with grayish striations throughout. The papillæ were hyperemic, and several of them showed to the naked eye what appeared as gray plaques somewhat similar to those found at autopsy of the human papillæ (30, 31, 32). No stones were present in the pelvis of the kidney nor on the papillæ of the kidney. The ureters were normal and contained no stones. The observations of the microscopic section are reported here as representative of all the dogs seen in Tables I and II which showed calcium to be present. They are as follows: The sections showed cloudy swelling to be present. There was marked shedding of the tubular epithelium in some places. Also, sections of the tubules, in which some of the cells were partly detached, contained varying amounts of calcium deposits forming solid dark blue casts, a greater quantity being in the detached and most abnormal cells. There were also necrotic cells present in some of the tubules. In another section the tubules presented a peculiar appearance showing evidence of having undergone atrophic changes. This might be explained as being a

Fig. 17. Kidney of dog cleared by modified Schultze method and stained with alizarin, a specific stain for calcium. The calcium deposits throughout the entire kidney are clearly shown in the darker areas. ×1.7.

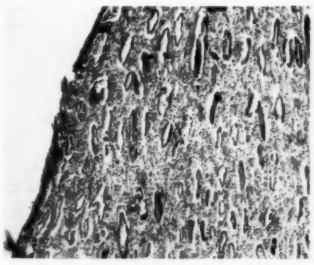

Fig. 18

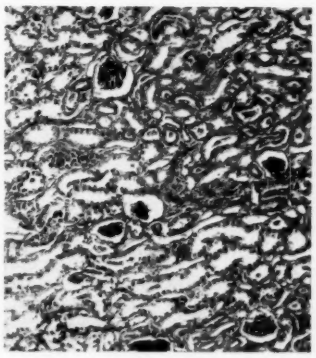

Fig. 19

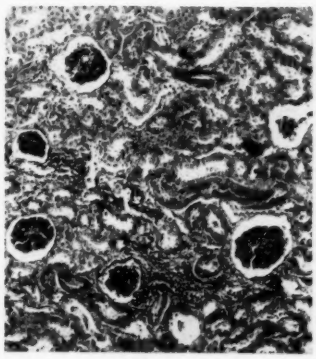

Fig. 20

Fig. 18. Photomicrograph of kidney of dog, showing calcium plaque on the side of a papilla. This is the second time that such a lesion has been observed in a lower animal. ×35.

Fig. 19. Photomicrograph of a section removed from the kidney of a dog. This section shows tubular degeneration, cloudy swelling, and degeneration of the glomerular tufts. ×60.

Fig. 20. Photomicrograph of section of kidney of dog, showing cloudy swelling, tubular degeneration, and calcification of Bowman's capsule, with glomerular tuft lying free in space. ×75.

precursor of tubular destruction before calcium is deposited (Figs. 14, 15, 16).

An interesting observation, and the second time ever reported, is the formation of a calcium plaque on the side of a papilla (Fig. 18). This occurred twice in 31 dogs under study. (Dog 97 reported in an earlier paper (3) and dog 294.)

The capillaries in the glomeruli were hyperemic, and hemorrhages throughout the sections were observed. Round cell infiltrations commonly were seen. The glomerular tuft appeared to be detached and shrunken, lying free in the glomerular space, and Bowman's capsule was calcified (Figs. 19 and 20).

One of the kidneys of each of dogs 43, 45, 47, 163, and 148 in the second group, instead of being sectioned, was fixed and cleared by the modified Schultze method and stained with alizarin. Figure 17 clearly demonstrates the heavy calcium deposits throughout the entire kidney, resembling a roentgenogram frequently observed in cases of nephrocalcinosis. I believe that this method of staining for calcium might be a more sensitive test than is the one usually followed.

SUMMARY

1. Thirty-one dogs were injected with parathormone subcutaneously to produce experimental hyperparathyroidism.

2. Viosterol was added in gradually increasing doses to the diets of 20 of these dogs, and exerted no observable change over those to which no viosterol was given.

3. The rise and fall of the serum calcium in all of the animals is in accord with the findings in similar experiments by others.

4. No bone changes were demonstrable in any of the dogs.

5. Vesical stone was present upon 3 occasions.

6. At autopsy the following were noticed: (a) omental and gastric hemorrhage, (b) visceral engorgement, and (c) marked renal damage.

7. Calcium plaques described by Randall were present in 2 dogs.

8. Almost all of the dogs showed deposits of intratubular calcium microscopically.

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CLINICAL SURGERY

FROM THE JAMES BUCHANAN BRADY UROLOGICAL INSTITUTE

RADICAL PERINEAL PROSTATECTOMY

Modification of Closure

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THE rare occurrence of shock, visual control of hemorrhage, and dependent drainage with the bladder completely at rest are definite indications for perineal removal of the enlarged prostate in many cases. One of the most important indications for perineal exposure and treatment of the prostate is the presence of early carcinoma. In certain cases of carcinoma of the prostate the lesion is confined within the capsule and fascias of the gland thus making the situation favorable for complete removal and cure of an otherwise surely fatal condition. The origin and development of the radical perineal operation for prostatic carcinoma by Young has been the only hope of cure in carcinoma of the prostate. The results of such operations in this clinic, in which many patients have been cured and are living and well today, can be found in the publications by Young. If the operation is carried out according to the accepted principles, the average case does not present unusual technical difficulties, especially when the carcinoma is confined within the capsule and is not accompanied by considerable benign enlargement.

Certain cases are encountered, however, in which there is either considerable benign enlargement or else extension of the carcinoma toward the apex of the gland so as actually to invade the beginning of the membranous urethra. When the latter has occurred, more extensive removal of the prostate is necessary and, in order to effect a complete cure, it is necessary to remove a portion of the external sphincter, which sometimes results in incontinence. Such a more extensive removal of the membranous urethra has at times presented difficulties concerning anastomosis of the bladder with the membranous urethra and triangular ligament. This has usually been carried out by means of sutures between the cut margin of the bladder to include the triangular ligament. These sutures have included the muscle mass which comprises the external sphincter. It occurred to the writer that such sutures which hold the bladder margin against the triangular ligament must pass through some portion of the external sphincter and when tied sufficiently tight to hold the bladder firmly in approximation they must ligate a portion of the sphincter. It is possible that this partial ligation of the external sphincter might account for the resulting weakness in some cases and especially even in cases in which no part of the sphincter has been deliberately excised or injured. For this reason the writer has devised the so called "traction sutures" shown in Figure 1.

Lalor fellow in Urology.

TECHNIQUE

The complete technique of removal has been described by Young and need not be reiterated here. Figure 2 shows the terminal views of the operation in which the apex of the prostate has been divided and the entire prostate freed, with complete separation at the vesical orifice. In this figure the trigone is being pushed backward from the seminal vesicles and their enveloping fascia, separating them from the base of the bladder. It also shows the division and ligation of the fascia and attachments at the tips of the seminal vesicles just before removal of the prostate, its enveloping fascia, and seminal vesicles *in toto*.

Section 1 of Figure 1 shows the perineal view of the open bladder after complete removal of the prostate, its fascias, the seminal vesicles, ampullæ, and distal vasa, for carcinoma of the prostate. The opening into the triangular ligament which comprises the urethra can be seen. The trigone and ureteral orifices are visible just behind the divided posterior lip of the bladder. Three traction sutures labeled *1*, *2*, and *3*, which have been placed in correct positions, are shown in this view. The first suture has included the anterior

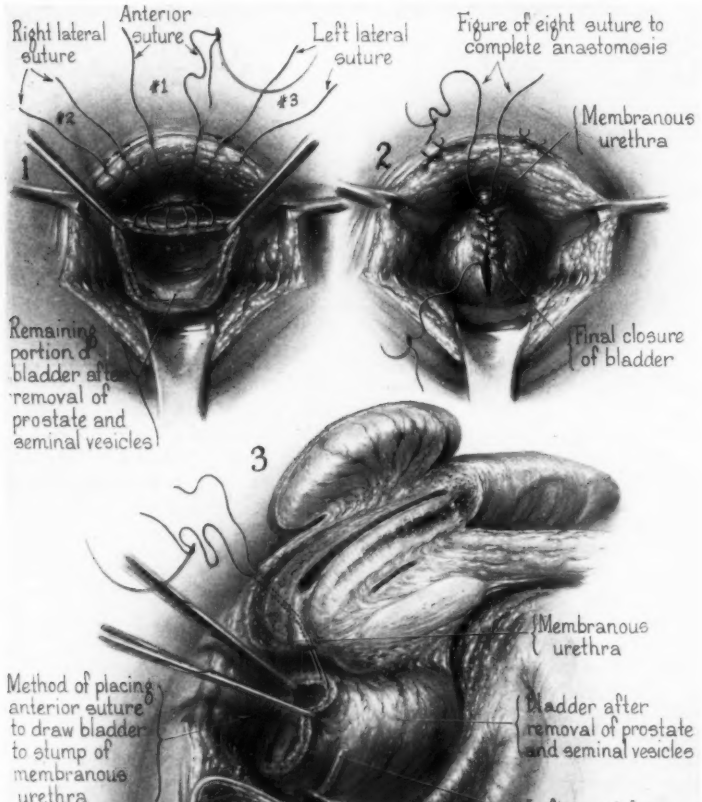

Fig. 1. Technique of traction sutures to avoid injury to external sphincter following radical perineal prostatectomy.

margin of the bladder in the midline by means of a firm bite so that the sutures emerge just at the margin of the bladder mucous membrane. This, *1*, or anterior suture, after leaving the bladder margin transverses the external sphincter on either side of the urethra just beneath the mucosa. It continues just beneath the mucosa through the external sphincter area, on through the triangular ligament and through a portion of the bulb of the urethra, perineal fat, and subcutaneous tissue so that it finally emerges on either side of the midline just beneath the skin at the anterior margin of the perineal incision. A sagittal view of this suture is easily seen in Section 3. It can be readily understood how traction on this suture will pull the anterior margin of the bladder from deep in the wound into true approximation to the cut margin of the membranous urethra. This is merely a straight pull through the area of the external sphincter and in no way ligates, constricts, or interferes with the function of this muscle as is conceivable heretofore by the use of conventional sutures. In Section 1 of Figure 1 two additional sutures designated *2* and *3* are placed well to the side of the midline and carried just beneath the mucosa to each lateral side of the membranous urethra and through the sphincter, being brought out through the perineum at the margin of the

skin incision well to each side of suture *1*. When traction is made on these three sutures and they are tied, considerable circumference of the cut bladder margin is drawn tightly against the membranous urethra and anchored there without any interference or constriction of the sphincter muscle in this area. The lateral sutures are usually tied first and then a strand of the anterior suture is tied to each of the lateral sutures so that there is no constriction across the midline. No. *2* chromic catgut is used so that the bladder is anchored in position until thorough healing of the divided mucous membrane can take place. It is more convenient to place these sutures with a urethral catheter emerging from the opening end of the membranous urethra, but the catheter has been omitted in these illustrations in order that a more clear idea of this suture principle can be obtained. Following the above procedure closure of the remaining open bladder is carried out as shown in Section 2 of Figure 1. When this final suture is completed to close the bladder opening there is usually a small opening at the point of anastomosis because the three traction sutures include only three-fourths of the circumference of the membranous urethra. A simple figure-of-eight suture carried in a very shallow manner into the triangular ligament and mucous membrane at this point is then placed to complete closure as is shown in Section 2. Complete closure is carried out over a moderate size urethral catheter in place to insure adequate drainage. The perineal wound is then easily and quickly closed in the usual manner with two small Penrose drains from each angle to insure drainage of extravesical tissues.

The writer has used the above traction sutures in only 4 cases but with entirely satisfactory results in each. The method is presented purely for what it is worth in the hopes that it will eliminate additional trauma to the muscles of the external sphincter following such a valuable procedure as total prostatectomy to cure carcinoma of the prostate.

SUMMARY

Certain cases of carcinoma of the prostate are confined within the capsule and fascias of the gland resulting in a favorable condition for radical removal and cure of an otherwise surely fatal disease. Statistics show an apparent cure in over 50 per cent of the cases followed over 5 years after Young's radical operation. The average case does not present unusual technical difficulties. In some instances it is necessary partially to excise and thus to injure the area of the triangular ligament comprising the external sphincter, this leads to incontinence. In other instances the sphincter is injured by ligation as a result of conventional sutures which are used to approximate the bladder margins to the membranous urethra.

The introduction of "traction sutures" to approximate the bladder to the membranous urethra following radical removal of the prostate for carcinoma has been presented here. These sutures pull the bladder firmly against the triangular ligament without any ligation of the musculature comprising the external sphincter and in this way minimize the chances of injury to this muscle with resulting weakness and partial incontinence. This procedure has been found valuable to the author in 4 cases, and it is believed that it deserves wider use.

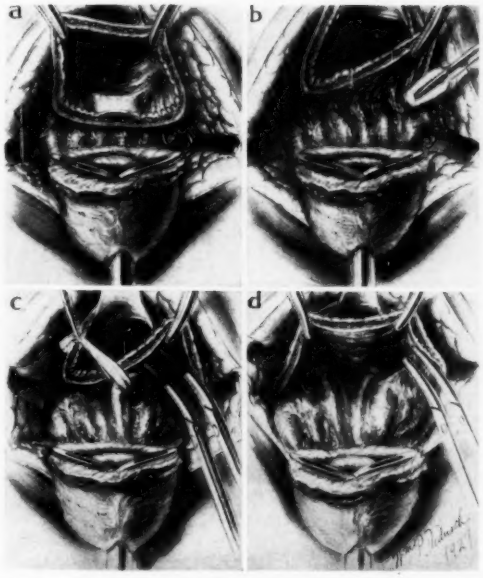

Fig. 2. Removal of entire prostate confined within its capsule, and also of seminal vesicles. a, Trigone has been divided and, b, bladder is being stripped up from vesicles and vasa, which are being ligated in c. d, Vasa have been divided and ligated, and fascial attachments of seminal vesicles are being divided, following which prostate, seminal vesicles, and terminal vasa are removed *in toto*.

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BRACHIOTHORACIC ADHESIONS

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BRACHIOTHORACIC lesions although not common are constantly occurring, and in almost any general hospital one or two cases a year may be seen. Since they are infrequent, the surgical attendant as a rule is not thoroughly familiar with a plan of treatment and directs his efforts along lines of general surgical principles. While doing this he not only is defeating his purpose but is allowing a vicious condition to develop that will increase the work to be done if not permanently injure the patient. In specialized institutions too where such deformities may be referred for treatment, there has not always been unanimity of opinion as to how such cases should be managed. In recent years, the works of Davis, Koch, and Blair and Brown have served to stimulate thought and discussion of the subject.

We may define brachiothoracic adhesions as any abnormal surface attachment between the arm and chest wall. It may be a congenital deformity, or it may be the result of violent traumatic injury or of an extensive inflammatory condition. It may follow an operative procedure involving the area or it may ensue from a severe burn. The latter is the most common cause.

The condition assumes importance out of proportion to its appearance. In the majority of cases the arm is practically incapacitated, the growth and development of the entire upper extremity, and, in marked cases, the growth of the mamma and structures of the chest wall are affected.

When a study of the production of such adhesions is made consideration should be given first to the anatomy involved. Anteriorly and medially, the pectoral and subclavius muscles exert a pull upon the arm that is directed toward the body. Posteriorly and medially, the same action is performed by the teres minor, teres major, and latissimus dorsi muscles. Between these two groups of muscles and between the chest wall and the arm lie the superficial and the deep fascias with the combined fascia at the apex forming the costa coracoid membrane. Second, familiarity with the pathological manifestations in the structures involved should be acquired. The loss of epithelium and its failure to regenerate completely over the arm, axilla, or chest wall, is the primary pathological process.

To repair this, there are two separate actions upon the part of the body: first, the adduction of the arm, thus protecting the important axillary structures and at the same time decreasing the amount of unepithelialized surface; second, the uniting of tissue as the granulation tissue surfaces come into contact with each other. Gradually the tissues become more organized, more fibrous, and finally a dense, hard, contracting web scar is formed.

With this understanding of conditions in mind, we approach the treatment of the patient.

First, the prophylactic phase should be discussed since it would seem from experience that this has been wrongly emphasized. In the medical journals and the surgical textbooks, many authorities advocate splinting, casts, and appliances to prevent the development of adhesions. When I started caring for patients with these adhesions I followed along such precepts. However, as I daily watched their suffering; as I saw contractures develop before my eyes, in spite of the splinting; as I followed patients in the out-patient department and observed the contractures increase, or develop in cases in which healing had taken place and in which patients at first appeared not to have an appreciable deformity; and finally as I operated upon such patients and demonstrated grossly and microscopically the amount and extent of scar tissue, I became convinced that this method of treatment was not the one most satisfactory (Figs. 1, 2, 3). The more I watched these patients, the more it became apparent that our efforts were misdirected. It is a fundamental principle of surgery that living tissue responds to injury in a well proved, definite manner. There is first a cellular exudate, the formation of a fibrinous framework, and soon among the cellular elements fibroblasts appear and effect the repair needed. Now, if the part is continually irritated, that is by stretching or splinting or by repeated painful dressings, there will be a greater production of fibrous tissue. As this fibrous tissue becomes more organized it shrinks and thereby causes contractures. This ultimate result may not show its maximum effect until perhaps 6 months after healing has taken place (Figs. 4 and 5).

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Fig. 1

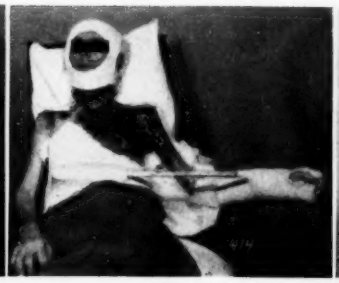

Fig. 2

Fig. 3

Figs. 1 and 2. Case No. 72041. Extensive infected burn with ulceration of neck, anterior chest, shoulder, arm, and left axilla. The parts are held apart with a plaster reinforced cast in as near the position of function as possible to maintain them. The patient required large doses of sedatives, the ulcerations showed very little signs of healing and it was impossible to eradicate the infection entirely.

Fig. 3. Case No. 72041. Result obtained by removing cast, applying moist saline packs and later application of skin grafts.

Fig. 4

Fig. 4. Case No. 373. Splinting may be used cautiously with children but should be of short duration and provide easy access for dressings.

Fig. 5

Fig. 5. Case No. 373. Grade I contracture of axilla and cord across cubital fossa. Treated early with grafts but shows effect of irritation by stretching resulting from cast.

Fig. 6. Case No. 66913. Ideal position and care for prevention of axillary adhesions.

Fig. 7, left. Case No. 66913. Result 4 months after Figure 6; not yet completely healed.

Fig. 8. Same case. Result 1 year later with Grade I contracture of left axilla. This patient had no splinting at any time.

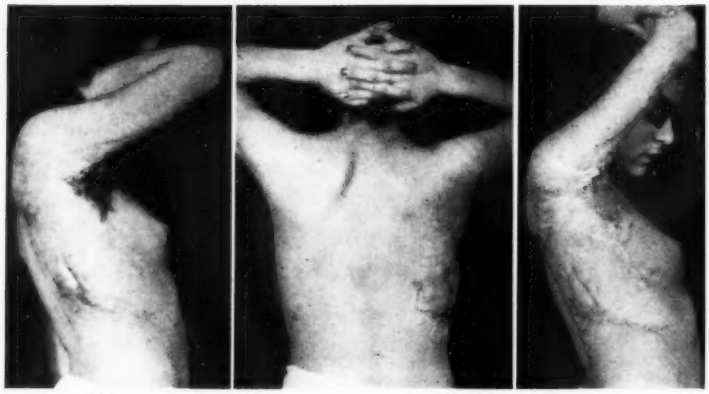

Fig. 9 Fig. 10

Fig. 9. Case No. 94880. Patient suffered an axillary adhesion following a burn 12 years ago. Five months ago she had been operated upon elsewhere with partial relief but an open infected ulceration remained.

Fig. 10. Case No. 94880. Result after clearing up of infection and skin grafting.

What treatment should be used to minimize if not wholly prevent axillary webbing? The process which we have found produces webbing must be reversed. That is, the patient must be placed in a position that is compatible with comfort (Fig. 6) yet allows attention to be given his wounds; and *at the earliest possible moment, the exposed surfaces must be covered over with skin grafts,* thereby permitting repair with a minimum of scar tissue (Figs. 7 and 8). Such treatment must be evaluated by the medical attendant; if, in his judgment, the patient's condition does not warrant it and if, in an effort to save life, axillary adhesions do result, the physician should not be condemned. On the other hand, a long, drawn out convalescence with innumerable painful dressings in an endeavor to allow nature to close in a large ulcer is most certainly open to criticism (Figs. 9 and 10).

When a case of axillary webbing in which the patient has recovered from the acute condition, presents itself for treatment, how should it be handled? In choosing the proper treatment for each case, many factors should be considered, as for instance the age and general condition of the patient, the availability of material with which to work, and the severity and extent of the disability. John Staige Davis has very concisely stated the problem: "The object of the treatment is primarily the restoration of the function of the arm, with the relief of the deformity and the prevention of recurrence. The successful recon-

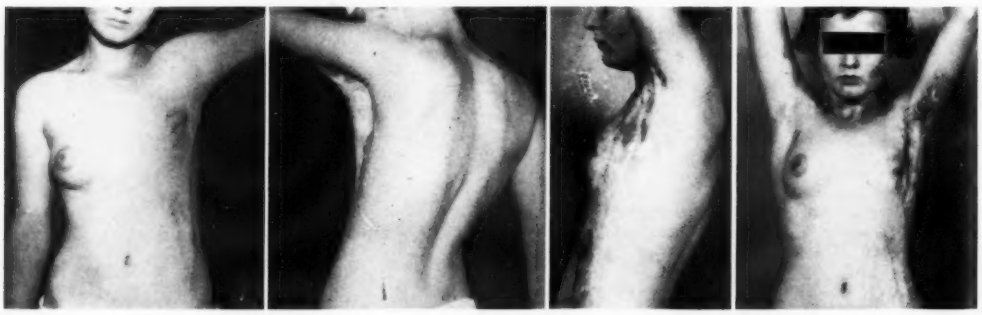

Fig. 11 Fig. 12

Fig. 11. Case No. 75158. Axillary adhesion of 12 years' duration.

Fig. 12. Case No. 75158. Result after Z plasty operation was done.

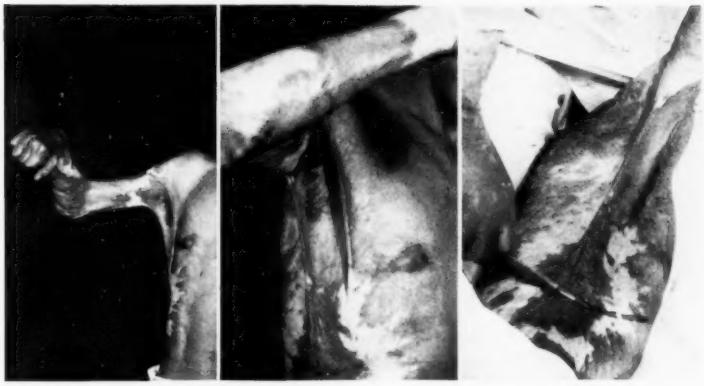

Fig. 13 Fig. 14

Fig. 13. Case No. 91533. Grade III contracture of axilla from old burn. This type of contracture is best treated by surgical attack at the point of maximum contracture as shown in Figure 14.

Fig. 14. Case No. 91533. Axilla and chest wall have been cleaned and prepared. The site of incision has been determined and is graphically shown.

struction or readjusting of the axillary space is the key to the relief of the condition."

I have found that the essential task to be effected, irrespective of the procedure, is either the complete removal of the cicatricial tissue or an interruption of its vicious line of pull. I have never had to resort to muscle sectioning or tendon lengthening, but I have relied upon secondary operations, carefully directed postoperative physical therapy, massage, and growth (Figs. 11 and 12).

A recent review and study of 29 cases in which patients were treated during the past 7 years brought forth the following pertinent facts:

1. There were 5 patients, 21 years or older. In 1 the adhesions resulted from surgery, the 4 remaining followed burns, and of these, 3 patients were burned as a result of epileptic seizures.

2. Excluding the adults, who represented 18.2 per cent of the total, the average age was 8.7 years, a fact which shows that this is a condition most often found in children.

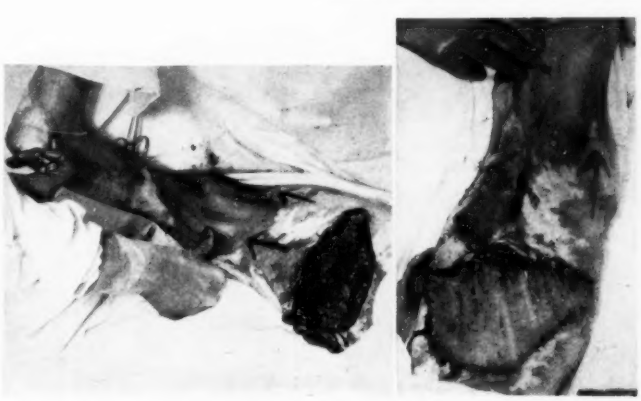

Fig. 15, left. The skin over the axillary folds has been under-cut and pushed upward to normal position, thus leaving an epithelial defect low on lateral chest wall.

Fig. 16. Case No. 91533. Axilla restored to normal appearance and open area on chest wall covered with a split graft.

Fig. 17, left. Case No. 42238. Grade II contracture in 16 year old boy resulting after surgical correction at age of 4 years.
Fig. 18. Correction by combination of Z plasty and low transverse skin graft.

3. There were 18 females and 11 males.

4. In 20 cases the adhesions followed burns from open fires.

5. The incidence of types showed grade II contractures, 14; grade III, 9; grade I, 4; and grade IV, 2.

6. The type of operation used was (a) Z plasty, 15 cases; (b) Z plasty and skin graft, 5 cases; (c) tube graft, 1 case; and (d) transverse incision and split graft, 8 cases.

7. Analysis of secondary operations showed: (a) 11 patients who had had a Z plasty operation alone required secondary operations; (b) 2 patients returned for further work after a Z plasty with skin graft; (c) of the transverse incision cases, only 2 patients required further work and this because the operation was done in stages.

8. Follow-up check-up gave the following results:

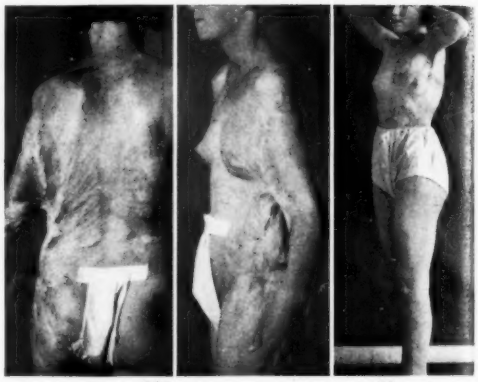

Fig. 19 Fig. 20

Fig. 19. Case No. 51795. Grade IV axillary web of 2 years' duration.
Fig. 20. Case No. 51795. Final result from four operations using Z plasty and transverse incision and skin grafts.

a. There were 8 patients operated upon 1 year ago, of these 2 had normal function and good anatomical reconstruction; 2 had good anatomical result with 25 per cent limitation of function; 1 had a small web postaxillary fold with 25 per cent limitation of function; 1 had a good anatomical result with 20 per cent limitation of function; 1 had a small web in the apex with 20 per cent limitation of function; and 1 had webbing of the posterior fold and 50 per cent limitation of function. I feel that these will improve with time and use of the parts. At a later date they can be further surgically corrected.

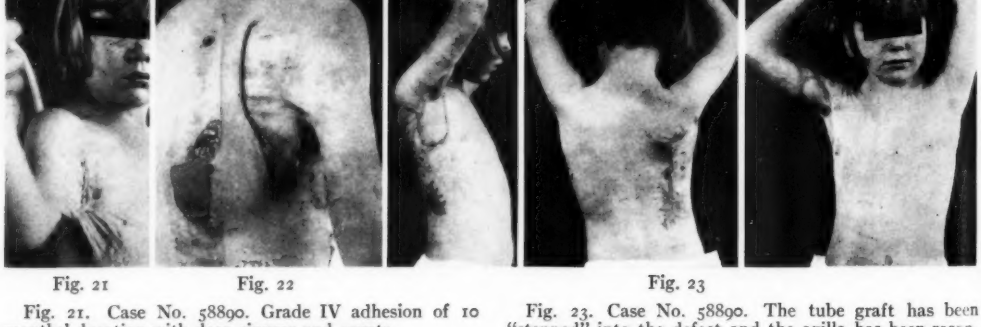

Fig. 21 Fig. 22 Fig. 23

Fig. 21. Case No. 58890. Grade IV adhesion of 10 months' duration with deep sinuses and crypts.
Fig. 22. Tube graft in process of being made.
Fig. 23. Case No. 58890. The tube graft has been "stepped" into the defect and the axilla has been reconstructed. Note restoration of function.

Fig. 24

Fig. 24. Case No. 72164. Grade III contracture of 10 months' duration.

Fig. 25

Fig. 25. Case No. 72164. Result 12 months after Z plasty operation and grafting ulcer on chest wall.

b. One patient operated upon 18 months previously has a good anatomical result with normal function.

c. There were 7 patients operated upon 2 years previously and of these 4 have good anatomical and functional results; 1 has a small web in the posterior fold that has excellent function; 1 has a good anatomical result with 10 to 20 per cent limitation of function; 1 patient with both axillæ involved shows limitation of function of 20 per cent in right and limitation of function of 40 per cent in left side.

d. There were 2 cases in the 3 year follow up and both showed good anatomical restoration and 100 per cent function.

e. In the 4 year follow up there were 2 cases. Both had good anatomical and functional results.

f. In the 5 year follow up there were 3 cases. One patient had a good anatomical and functional result, and 2 showed good functional results with small webs in the posterior axillary folds.

g. There was 1 patient in the 7 year follow up. This patient has a good anatomical and perfect functional result.

PLAN OF TREATMENT

As a result of the experience gained and the results obtained, the following plan of treatment has been evolved:

Age. I feel that unless there is marked deformity, that is a contracture of grade IV or more, no surgical treatment should be done until the child is 3 to 4 years of age.

Preparation. A thorough check up of the patient's general condition should always be made and any variations from normal corrected. Should a patient show an anemia, his operation is postponed until the condition is relieved. Any small ulcerations on the webs are healed. Daily cleansing and preparing of the skin is carried out until the skin appears healthy. Small sinuses, crypts, and pockets are thoroughly cleaned out. Ultra violet treatments or direct exposure to sunlight are quite often given.

Operation. The exact procedure will naturally depend upon the grade of contracture and the quality of the skin available.

In grade I contractures, I have found the following to give the best results. A transverse incision, usually 5 to 6 inches in length, is made completely through the cicatricial tissue down to muscle at a level of 4 to 6 inches above the crest of the ilium, thereby permitting the arm to be fully extended. A diamond-shaped defect is thus created on the lateral side of the chest wall and lumbar region which can easily be covered with a split graft.

This procedure not only corrects the contracture but does so with very little disturbance of the axilla (Figs. 13 to 16).

In grade II contractures, if the webs are loose and of good blood supply, a Z plasty alone is done. If deformity is not completely corrected after the Z plasty, a transverse incision is made and a skin graft is applied (Figs. 17 and 18).

If the webs are dense, 2 or more operations of the transverse type, thus raising the arm in stages, are performed. This technique tends to soften the webs by releasing the pull and allows them to assume the shape of the axillary folds as they are pushed upward (Figs. 19 and 20).

In grade III and grade IV contractures the same technique as in grade II may be used but more stages are required. If the contractures are too dense to be mobilized, two methods of procedure may be followed.

A tube graft elsewhere on the body may be formed to furnish the tissues for the reconstruction of the axilla and "stepped" into position (Figs. 21 to 23).

A plastic operation, which would free the contractures, permit the arm to be raised, and

cover the defect with a split graft, is carried out and may be accomplished in 2 or more stages.

Splints, supports and dressings. The arm is elevated to the full amount of correction accomplished by the operation and compatible with good circulation of the forearm and the hand. This elevation must be maintained until the period of healing is ended, usually 10 to 14 days.

When the patient is 3 to 6 years of age and difficult to keep quiet, we apply a sea sponge pressure dressing to the axilla and then put on a plaster shoulder spica. This is bivalved and the parts are dressed at the end of 7 to 9 days. The cast is then worn for another 7 to 10 days, as deemed necessary.

In older patients dressings are applied in the same manner but a cast is not worn. A thick pillow covered with rubber sheeting makes an excellent support, is more comfortable, and lends itself to changes, and can as well be completely removed. It is not necessary for these patients to wear supports as long as it is for the younger patients.

Postoperative care. These patients are given ample sedatives for 24 hours; then, depending upon the individual requirements of each patient, mild sedation to the point of definitely restricting activity for another 48 hours is carried out. Following this, patients are allowed bed movement but are not allowed up until fully 8 days after operation.

When they are allowed up, passive movements as well as gentle massage can be instituted. At the end of 10 days, active movements are started, and from then on encouraged, but the individual is ever cautioned not to overstretch or injure the parts. We have found loom weaving, wheel exercises, and swimming to be excellent aids to restoring function.

CONCLUSIONS

1. The condition of brachiothoracic adhesions is a natural consequence of the physiological healing of two adjoining body surfaces in which there has been extensive epithelial loss.

2. Such lesions can be minimized, if not wholly prevented, by proper position, well directed dressings, and early skin grafting. The use of painful splints or casts is of no real value and usually results in aggravation of the existing condition.

3. In the treatment of severe burns, when there is extreme shock, pain, and loss of tissue, it would seem that the decision to allow an arm to adhere to the chest wall to facilitate closing in of a large open area and at the same time to afford protection to vital structures of the arm and forearm, is surgically sound.

4. Axillary adhesions should not be treated by means of traction, stretching, or the wearing of appliances but by a definite surgical procedure. The problem of treatment is predicated upon the restoration of the arm, axilla, and chest wall, to a condition as near to normal in appearance and function as is compatible with available tissues.

5. Extensive surgery of muscles, ligaments, and joint capsules is rarely indicated.

6. Patients must be permitted to grow and must be given time to readjust themselves before a final decision as to results is made (Figs. 24 and 25).

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SVANTE ORELL, Stockholm, Sweden

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Cases in the last group seem to offer the best possibilities of good results.

Eden, in Jena in 1917, was the first to use bone grafts for the treatment of habitual dislocation of the shoulder. His method consisted of transplanting a piece of the tibia to the anterior rim of the glenoid cavity so that the graft projected at least 10 millimeters from the rim, and then fastening it with periosteal sutures or with pegs under the raised periosteum of the neck of the scapula.

Only 10 days after Eden, Hybbinette performed a similar operation in Stockholm. He made a wide opening in the joint to secure good exposure and to permit observation of the articular changes in detail. At first he used a tibial graft. Later, however, due to observations made during operative procedures, he took a transplant from the iliac crest and inserted it into a periosteal pocket in the anterior lip of the glenoid cavity by means of a specially designed instrument. The transplant was provided with an edge to rest against the rim of the cavity, to prevent the graft from slipping too far into the periosteal pocket. The free edge of the transplant was made to project 3 to 4 millimeters in front of the rim of the socket. With this method Hybbinette obtained remarkably good results.

Subsequently, Oudard attached a free bone transplant obtained by chiselling obliquely through the coracoid process to the anterior glenoid margin. In 1924, Noesske modified Oudard's method. He chiselled obliquely into the coracoid process and bent down the flexible piece of bone so obtained toward the anterior rim of the cavity where he attached it. He thus used a sort of pedicled graft.

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Since it is so important that the implanted bone be well attached to the scapula, I shall now describe a method of operation which resembles that of Speed and which I used in a case of habitual dislocation of the shoulder. This technique provides remarkably good immediate fixation of the bone implant. The procedure is simple and time-saving.

CASE REPORT

A married woman of 34 years had suffered from recurrent dislocation of the right shoulder since 1932. One day she fell down and knocked her shoulder out of joint, but she had no difficulty in putting it back into place herself. After that, certain movements were painful, especially when she extended her arm, which caused a "twinge" in her shoulder. The dislocation recurred and gradually increased in frequency from once every few months to several times a week. She was always able to replace the head in the joint herself until November 28, 1938, when she found it necessary to consult a physician. From then on it was difficult to reduce the dislocation. She was admitted to Värnamo Hospital on January 13, 1938. Her shoulder had then been out of joint 3 hours and could not be replaced. After immediate reduction under anesthetic and fixation of the shoulder in a bandage, the patient insisted upon an operation.

Operation, consisting of bone implantation in the anterior rim of the scapula, was performed under evipal

cover the defect with a split graft, is carried out and may be accomplished in 2 or more stages.

Splints, supports and dressings. The arm is elevated to the full amount of correction accomplished by the operation and compatible with good circulation of the forearm and the hand. This elevation must be maintained until the period of healing is ended, usually 10 to 14 days.

When the patient is 3 to 6 years of age and difficult to keep quiet, we apply a sea sponge pressure dressing to the axilla and then put on a plaster shoulder spica. This is bivalved and the parts are dressed at the end of 7 to 9 days. The cast is then worn for another 7 to 10 days, as deemed necessary.

In older patients dressings are applied in the same manner but a cast is not worn. A thick pillow covered with rubber sheeting makes an excellent support, is more comfortable, and lends itself to changes, and can as well be completely removed. It is not necessary for these patients to wear supports as long as it is for the younger patients.

Postoperative care. These patients are given ample sedatives for 24 hours; then, depending upon the individual requirements of each patient, mild sedation to the point of definitely restricting activity for another 48 hours is carried out. Following this, patients are allowed bed movement but are not allowed up until fully 8 days after operation.

When they are allowed up, passive movements as well as gentle massage can be instituted. At the end of 10 days, active movements are started, and from then on encouraged, but the individual is ever cautioned not to overstretch or injure the parts. We have found loom weaving, wheel exercises, and swimming to be excellent aids to restoring function.

CONCLUSIONS

1. The condition of brachiothoracic adhesions is a natural consequence of the physiological healing of two adjoining body surfaces in which there has been extensive epithelial loss.

2. Such lesions can be minimized, if not wholly prevented, by proper position, well directed dressings, and early skin grafting. The use of painful splints or casts is of no real value and usually results in aggravation of the existing condition.

3. In the treatment of severe burns, when there is extreme shock, pain, and loss of tissue, it would seem that the decision to allow an arm to adhere to the chest wall to facilitate closing in of a large open area and at the same time to afford protection to vital structures of the arm and forearm, is surgically sound.

4. Axillary adhesions should not be treated by means of traction, stretching, or the wearing of appliances but by a definite surgical procedure. The problem of treatment is predicated upon the restoration of the arm, axilla, and chest wall, to a condition as near to normal in appearance and function as is compatible with available tissues.

5. Extensive surgery of muscles, ligaments, and joint capsules is rarely indicated.

6. Patients must be permitted to grow and must be given time to readjust themselves before a final decision as to results is made (Figs. 24 and 25).

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Fig. 1. a, Osteoperforator; b, instrument to assist the extraction of osteoperforator; c and d, pointed, arched os purum transplant, the convex wall of which consists of compact bone and the concave wall of spongy bone; e, bakelite handle which may be used to help drive in the os purum graft; f, operative mallet.

anesthesia on January 15, 1939 (Orell). An incision was made from the coracoid process down along the arm between the pectoralis major and deltoid muscles. The joint capsule was divided in the anterior part of the joint, and a finger was inserted for palpation of the rim of the glenoid cavity. The extracapsular soft parts, including the subscapularis muscle, were bluntly divided all the way to the anterior edge of the glenoid cavity. Then the periosteum was loosened from the scapula and a specially designed osteoperforator (Fig. 1, a) of the same shape as a pointed lamella of os purum (Fig. 1, c) was driven into the scapular bone immediately medial to the anterior edge of the glenoid cavity. After the osteoperforator was removed with the help of a special instrument (Fig. 1, b), an os purum lamella was driven extracapsularly into the cleft until it was securely in place. Most of the os purum graft was nipped off to leave only the amount required to prevent the head from slipping past the glenoid cavity's anterior edge. The wound was sutured in layers and the arm was fixed in an ordinary bandage.

Healing was primary. During the first 2 weeks after operation the arm was kept in adduction. For the next 2 weeks the patient performed exercises under the direction of a gymnast, after which she returned home and did the exercises by herself.

When the patient came back for re-examination on July 30, 1939, she stated that since the operation, in January, 1939, she had never experienced any of the old pain or trouble. The joint felt stable, and in spite of the fact that since her discharge in February she had gone back to doing all her own housework, she has had no dislocation. The muscles in the right shoulder were just as strong as those in the left. There was no atrophy. The strength of both arms appeared to be equal. She could not lift the right arm quite as high as the left, but the difference was not great (Fig. 2). She was able to throw a baseball.

Fig. 2. The patient 6½ months after operation, standing with her arms lifted high (sketch taken from 16 millimeter film).

This operation is very simple and requires but a short time. Naturally, the osteoperforator must be manipulated so that its sharp point, which is controlled by the finger inserted through the hole made in the anterior wall of the joint capsule, does not slip but becomes fixed immediately in the bone. Nor should the scapular bone be cracked. Its tissue seems to be very homogeneous and finely meshed, so that there is no risk of cracking if the operative mallet is handled carefully and sensitively. When the osteoperforator has been removed, there seems to be no difficulty in driving in the similarly shaped os purum graft. Moreover, with the use of os purum one avoids the loss of time incurred in taking a bone transplant from another part of the body.

Insertion of a thin strip of bone extracapsularly in the anterior edge of the scapula appears to be sufficient to prevent recurrent dislocation. It has been said that the atrophy of the capsule after an operation following the method of Eden or Hybbinette is essential for a successful result, and that it is therefore very important that the arm be maintained in adduction for a long time after the operation. Such a step, however, involves a risk of considerable limitation of movement, which may later be difficult or impossible to eliminate. For this reason it seems to me that, if possible, the arm should not be fixed for more than 10 to 14 days after operation. With the aforementioned procedure, in which the atrophy of the capsule is clearly of minor importance, it should be possible

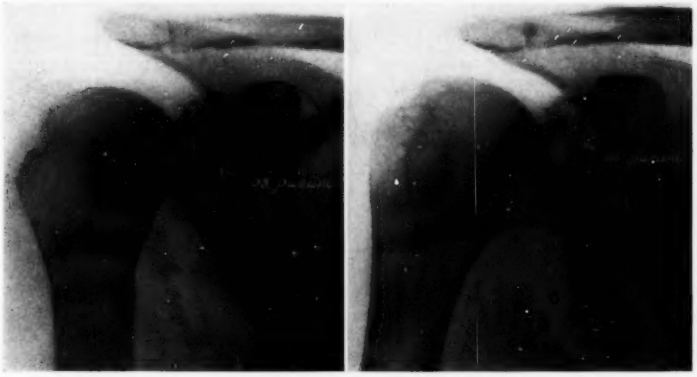

Fig. 3. Roentgenograms of the right shoulder joint. a, Left, 6 days after operation—the os purum transplant is seen in the anterior edge of the joint cavity. b, 222 days after operation—the os purum graft is largely resorbed. The joint cavity has a normal appearance.

to cut down the period of fixation of the arm to a still shorter time, since the inserted os purum graft fits tightly into the scapula, and as a result of the compression of the bony parts, the connective tissue in the spongiosa of the scapula has every chance of quickly growing into the canal system of the graft and "soldering" it by means of living tissue. Probably most of the os purum lamella will be resorbed, but enough should be reconstructed in new bone, due to the mechanical stress between the head of the humerus and the rim of the glenoid cavity, to prevent luxation (Fig. 3).

SUMMARY

A method of operation for habitual dislocation of the shoulder joint is described. It consists in making a cleft or fissure extracapsularly in the desired place in the rim of the glenoid cavity by means of a specially designed osteoperforator and inserting into it a pointed os purum transplant. The graft is nipped off so that it projects only slightly beyond the rim of the glenoid. Due to the fact that the cleft in the scapula and one side of the os purum lamella consist of spongy bone, a remarkably good immediate fixation between them is obtained. This is later completed by the bone-forming connective tissue from the scapula, gradually growing into the graft. This bone formation seems to be sufficient to prevent luxation, even after most of the os purum transplant has been resorbed. The postoperative fixation of the shoulder and the resulting stiffness of the joint can in this way be reduced to a minimum. The operative method is technically simple and time-saving, since the transplant is not taken from another part of the body.

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SPECIAL FEATURES IN THE MANAGEMENT OF SURGICALLY DIFFICULT MALIGNANT GROWTHS AND KINDRED LESIONS OF THE PELVIC VISCERA

ARTHUR H. CURTIS, M.D., F.A.C.S., Chicago, Illinois

AN intensive study of pelvic anatomy, both in the operating room and in anatomical dissections, has been carried on with Dr. Barry J. Anson.[1] Not only because of his originality in the pursuit of gross anatomy, but also because of his keen interest in the adaptation of what we have learned to the solution of clinical problems, I am particularly grateful to my co-worker.

PELVIC ANATOMY IN RELATION TO SOME DETAILS OF GYNECOLOGICAL DIAGNOSIS AND SURGICAL TECHNIQUE

Attention is directed to the cellular tissues of the pelvis, for the pathway of the lymphatics and blood vessels is entirely by way of these cellular tissues; therefore an exact knowledge of the latter will portray in detail the route by which infections spread and cancer invades. Cellular tissue is comprised of connective tissue cells and may serve as loose packing, as in the areolar tissue within the broad ligament; or it may be more dense, tense, and firm, thus constituting ligamentous tissue. Furthermore, the connective tissue fibrils may serve as a filler, or they may form a firm fascial covering or a thin, more or less incomplete sheet, which gradually fades away like the thin bark on a white birch tree.

In a recent publication concerned with cystocele, it was demonstrated that after removal of the intestines, peritoneum, and subserous tissue, the firm fascia which lines the muscles of the pelvic floor is reflected upward as a collar over each of the three hollow viscera, namely, the bladder, the vagina and uterus, and the rectum. The relatively thin collar around the bladder is in apposition with the heavier vagino-uterine collar, the bladder pillars constituting a connecting bridge.

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[1]Curtis, Arthur H., Anson, Barry J., and Beaton, Lindsay E. The anatomy of the subperitoneal tissues and ligamentous structures in relation to surgery of the female pelvic viscera. Surg., Gynec. & Obst., 1940, 70: 643–656.

More recent observations in the operating room correlated with anatomical dissections have demonstrated the continuity of the investing fascial collars with the supporting structures at a higher level. Proceeding from above downward, the fascial investment over the uterus first becomes distinct posteriorly slightly above the level of the uterine attachment of the uterosacral ligaments. Anteriorly, is noted a remarkably broad attachment of the round ligaments down to the level of the broad ligament branch of the uterine vein; immediately below this point the fascial collar becomes dissectable.

Lateralward, the uterovaginal and vesical fascial coverings become continuous with the Mackenrodt ligament, which spreads out, tent-like, to become inserted, fan-shaped, into the fascia overlying the obturator muscle and the muscles of the pelvic diaphragm. With the peritoneum removed one sees that Mackenrodt's ligament really begins above at the level of the obliquely coursing broad ligament branch of the uterine vein, and that there is a "core" consisting of this vein and the slightly more inferiorly placed uterine artery and accompanying vein, all enclosed in a vascular compartment; one notes also posteriorly, that the uterosacral ligament is continuous laterally with and really is an integral part of Mackenrodt's ligament, as well as continuous with the uterovaginal fascial collar. Upward and downward extensions of the fascial sheath surrounding the vessels constitute firm investments accompanying the ascending and vaginal branches of the uterine artery and vein.

The course of the ureters is a topic of never ending interest and concern, even to those most experienced. Noteworthy surgical landmarks therefore merit a few words. When the pelvis is entered the ureter is found to lie immediately medial to the attachment of the infundibulopelvic ligament at the pelvic brim. Therefore, in peritonization, after removal of the uterus together with the ovaries, the ureter is hazardously near, just beneath the peritoneum, and the peritonizing suture should never be placed deeply on the medial side without preliminary palpation of

the ureter. Located 4 centimeters from the uterus at the level of the internal os, the ureters are 1.5 centimeters lateral to the lowermost cervix at the point of their greatest proximity to the uterus; in their downward course they lie beneath the tent-like, fan-shaped, lateral extensions of Mackenrodt's ligaments, crossing under the uterine vessels, and are situated approximately 8 centimeters apart at the level where the pelvic surgeon usually places clamps on the vagina in performing a simple, complete hysterectomy. Another landmark is afforded by the bladder pillars; the ureters are located just anterior to and 1 centimeter lateral to the pillars. In the detail of radical hysterectomy for cancer it is helpful to know that the ureters enter the bladder obliquely convergent and are ensheathed in extensions of the musculofascial covering of the bladder. Also worthy of emphasis is the fact that a considerable fascial investment extends along the ureter, thus creating a pathway for lymphatic drainage from the cellular tissues contained within Mackenrodt's ligament.

All of this means that a new concept is available in appraisal of local extension of cancer beyond the confines of the uterus. The spread of cancer is inevitably through the cellular tissues along the fascial planes which have been depicted. With detailed knowledge[1] of the cellular anatomy of the female pelvis now available, local metastases may be expressed in terms of interstitial involvement, superseding our heretofore hazy understanding of lymphatic anatomy and lymphatic drainage. Palpation of the tissues in an attempt to evaluate the invasion of these anatomically definite routes of extension promises to yield more trustworthy deductions relative to the extent of the malignant process; likewise, evaluation of the condition of the cellular tissues as well as the glands encountered at operation should be preferable to attention concentrated on palpation of the lymph nodes.

The anatomy of the bladder in relation to pelvic surgery is intriguing. The musculofascial investment is fortunately somewhat thicker on the inferior surface and is greatly strengthened by the contiguous, very much thicker musculofascial collar of the vagina and cervix. At complete hysterectomy, however, we separate in the plane between the vesical and the cervicovaginal collars, where the vesical fascia is much attenuated, with the result that the integrity of the bladder wall is often endangered. This I have demonstrated repeatedly through the simple expedient of pre-operative instillation of 20 cubic centimeters of 1:1,000 methylene blue solution into the empty bladder as a safeguard: the blue solution shines through unduly denuded areas, warning of danger, and it spills in case of injury. In this connection, I am impelled to emphasize the frequency with which one invades the musculofascial envelope of the bladder in the routine of complete hysterectomy, and also the hazard of tissue forceps' injury and leakage from the bladder which doubtless is accountable for some of the cases of fistula heretofore ascribed to the too deep placement of sutures. The fascial wings over the bladder spread far laterally, and the bladder and ureter tend to fall farther laterally upon surgical dissection than one might surmise. Therefore, in the performance of a magnified Wertheim operation, zealous removal of all available cellular tissue in the deep pelvis lateral to the ureter is somewhat hazardous.

[1]Loc. cit.

MANAGEMENT OF CASES WITH (1) GROWTHS ENCROACHING UPON OR INVADING THE BLADDER, (2) INSEPARABLE WELDING OF THE BLADDER TO THE UTERUS, (3) INSEPARABLE OBLITERATING CUL-DE-SAC ADHESIONS OR TUMOROUS INVASION OF THE CUL-DE-SAC, (4) UNILATERAL EXTENSION OF MALIGNANT OR OTHER INVASIVE GROWTHS

With added experience the impression has been gained that with dissection along fascial planes in the removal of most genital pathological lesions, the tissues operated upon may be made to fall apart, and that thereafter they fall together readily in completing the operation. Such blunt dissection is inevitably more gentle, and tissues united without tension heal kindly with lessened distress during convalescence. This does not imply that work may be done without awareness of the fact that waste of time increases morbidity and mortality.

Cases of carcinoma of the body of the uterus, of the lower corpus or endocervix, and cases of pelvic endometriosis may involve the bladder sufficiently to make hysterectomy hazardous (Fig. 1). Those cases in which resection of the bladder is indicated will not be dwelt upon. In all other such instances the technique is essentially as simple as in the performance of an ordinary hysterectomy if one leaves the bladder undisturbed until the uterus is otherwise freed; then, with excellent visibility and perfect control, the uterus separates from the bladder without effort along natural lines of cleavage. With this procedure, as well as in those difficult adherent cases in which it appears expedient to separate the bladder according to the more usual technique, or perhaps with approach from the lateral aspect, methylene blue solution in the bladder is a safeguard.

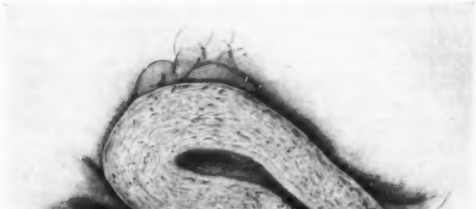

Fig. 1. Adenocarcinoma of lower uterine corpus and endocervix with invasion of wall of bladder.

Another group of cases to be considered are those with obliterating lesions of the cul-de-sac with welding of the rectum to the vagina and cervix. It is self-evident that cases of endometriosis in which both ovaries are to be removed require no cul-de-sac surgery. But if one ovary is to be spared, also in cases with other tumorous lesions or cellulitis of the cul-de-sac, dissection of this region may be indicated. Here, analogous to the procedure in cases with a firmly welded bladder, the cul-de-sac dissection may be reserved advantageously for the final step, which makes the technique less formidable and the hazard of rectal injury minimal.

Present day emphasis on simplification of the technique of complete hysterectomy by generous mobilization of the bladder and, particularly, by freeing the uterus posteriorly and posterolaterally, has militated against our appreciation of other procedures. In cases with firm fixation of the bladder and an immobile rectum, a complete hysterectomy can be accomplished from side to side with facility and with greater ease and less hazard; dissection of these viscera can be reserved for the final steps of the procedure with separation of the more inaccessible viscus last.

Among the reasons for the 50 per cent death rate from corpus cancer are (1) the tendency to tubal spill with extension to the ovary and peritoneum and invasion of the contiguous cellular tissues, and (2) extension of lower corpus growths either directly or via the endocervix into the lymphatics of the vascular compartment and cellular tissues of Mackenrodt's ligament. Such extensions are often patently unilateral; those from ovarian growths, tubal carcinoma, and endometriosis may also be unilateral. In many such cases the customary simple removal of the uterus and adnexa may be supplanted advantageously by unilateral, simple division of the tissues on the uninvaded side of the uterus, with exposure of the ureter and a Wertheim procedure on the side of greater invasion; a "magnified" Wertheim complete eradication of all removable structures may be resorted to in extreme cases.

A great many years ago there was smug complacency relative to the idea that removal of a major pelvic tumor mass often spares life for many years, despite failure to eradicate the growth in its entirety. Now it is known that extensive surgery with exposure of the ureter and removal of all available cellular structures, usually unilateral, sometimes bilateral, greatly increases the expectancy of selected patients of this group; even those with an extensive omental cake and implants which are not removed can be controlled by roentgen therapy.

CARCINOMA OF THE CERVIX

The operative treatment of carcinoma of the cervix will be omitted and only a summarized listing will be given of essential details in the non-operative management of this last and most important item among the subjects selected for discussion.

1. A well trained gynecologist should be able to diagnose carcinoma of the cervix from its gross appearance alone in nearly all instances. I have no brief against routine preliminary removal of fragments of tissue for biopsy study before venturing a diagnosis, but I do not often resort to this procedure. On the other hand, I frequently find that establishment of a certain diagnosis necessitates examination under anesthesia, often with instrumentation to provide adequate exposure and to determine the exact nature of atypical or hidden areas. I not only condone such examinations, but would emphasize the value of the study of cervical cancer patients when thus asleep, in order to evaluate the intricacies of the therapeutic problem which confront us.

At the time of the examination in the operating room it is my custom to state definitely, "It is," "It is not," or "I do not know whether it is cancer." Treatment is instituted immediately in nearly all instances and is delayed only in that small percentage of cases in which the diagnosis is in doubt and must await microscopical examination. Tissue is invariably taken for biopsy to make final confirmation of the diagnosis and to determine the type of growth.

2. In the event that no malignancy is grossly recognizable, which usually means that malignancy is unlikely, the diseased area should be removed *in toto* as a prophylactic and diagnostic measure, the entire excised tissue immediately sliced for gross inspection, and all of the several pieces so obtained imbedded for microscopical study of one or more sections from each. We rarely find it helpful to resort to the Schiller test or colposcopic examination, but believe we should remain somewhat open-minded about the latter.

3. Non-ulcerated, non-infected cases of cancer with a favorable prognosis (Fig. 2), i.e., stage I and stage II clean cases, are treated with a tandem of radium capsules within the canal together with radium needles imbedded in a palisade encircling the cervix. Some maintain, and probably with reason, that radium within the canal together with radium placed against the cervix is equally efficacious. Our usual dosage varies from 3,500 to 4,500 milligram hours. No further radium treatment is required for many months, at least, in these clean cases; but these patients should be seen frequently and the cervical canal kept well dilated.

4. In all cases of cervical cancer with a serious prognosis an intravenous pyelo-ureterogram is indicated, in order to determine whether there is involvement of the urinary tract.

5. Ulcerated or craterous, necrotic cancer must be healed on the surface before intensive radiation is used to avoid the danger of pelvic cellulitis. Surface healing may be accomplished with surgical diathermy or with a palliative radium application of 1,000 to 1,500 milligram hours, preferably in a bomb. Many weeks later, after healing of the necrotic surface, radium treatment may be given as described for non-infected, favorable cases.

Some of this group cannot be radiated adequately without endangering the bladder or ureters; in such instances, simple vaginal dissection readily places them out of harm's way. With the bladder safely anchored in an elevated position we may give intensive radium treatment with selective implantation of needles in the musculofascial parametrial tissues aided by radium capsules within the uterine canal.

After preliminary radium treatment has healed the surface, patients with deeply craterous cancer and others with relatively advanced lesions are treated conservatively with a radium bomb against the cervix. Painstaking care is essential in order to avoid destructive overtreatment. Patients with hopelessly extensive and deeply ulcerated cancer may receive no treatment other than roentgen therapy; but even this must be administered with caution, for the possibility of cure is slight and of doing harm is great.

Fig. 2. Typical squamous cell carcinoma of cervix.

6. Intensive high voltage roentgen therapy is instituted immediately in all cervical cancer cases as soon as a diagnosis is established, and the patient is carefully watched and guarded against the hazard of overtreatment which is considerable. Roentgen therapy is a great boon. But, as we learned some years ago, it is not innocuous; thus, in one bad case in which the patient was treated intensively, there was leucopenia, a clinical picture of agranulocytosis. This patient died with myriads of small ulcerations of the colon but not a vestige of cancer was discovered at autopsy.

7. And now for a suggestion to which many may not be receptive: Howard Kelly was right when he stated that 85 per cent of the value of radium therapy lies in the first treatment and 85 per cent of the harm which we do is ascribable to subsequent radiation. But that dictum, admirable with the knowledge and methods of early days of radiation therapy, is no longer applicable with our somewhat widened vision . . . . The extent of the cancerous lesion cannot be appraised accurately in the majority of serious cases at the time of the first treatment. Therefore, I have found it immeasurably helpful to make another painstaking study almost routinely under anesthesia, several months or a year or more subsequent to the first intensive treatment with radium. One can then determine with considerable precision and in detail what has been accomplished. Such an examination should, of course, be made with radium on hand and with instruments prepared for simple surgical intervention

Fig. 3. Carcinoma developed in a cervical stump.

to insure satisfactory placement of the radium.

The following is only one of many cases in point:

A patient was brought to us with a history of prolonged, supposedly functional bleeding. Palpation was negative, but a hidden cancer was suspected. Under anesthesia we found an endocervical malignancy, the cervix a craterous shell, the bladder resting on a thin film of cervix anteriorly, and the cervicovesical wall so thin that it felt like a fluctuant fetal sac. Tissue was obtained for histological study; with fear and trembling we gave 800 milligram hours of radium within the canal, then instituted roentgen therapy, and 3 weeks later applied a radium bomb to the cervix. After 4 months, examination under anesthesia revealed smoothly healed surfaces, the cervix a firm thin shell, anteriorly of tissue paper thinness. The bladder was mobilized by blunt dissection and shoved upward; the uterus was pulled downward so that the internal os became the external os, and then it was possible to treat with intensive radiation. The patient has remained free from evidence of cancer on repeated examinations under anesthesia.

8. Cancer of the cervical stump (Fig. 3) may be treated by radiation alone; but we prefer removal and radiation in those patients in whom the growth is limited apparently to the cervix; and we employ radiation followed by subsequent removal in all other cases except those which are hopelessly extensive. My advocacy of surgical removal, in preference to roentgen-radium radiation alone, is prompted by bad results from treatment solely with radiation therapy, also by the simplicity of vaginal removal of the cancerous stump, and by the fact that it is usually impossible to estimate the extent of the cancerous process in these cervical stump cases and therefore difficult to determine the amount of radiation required without exploration to reveal the stage of the disease. These views relative to the management of cancer of the cervical stump are still in a state of flux, subject to revision.

CONCLUSIONS

1. With detailed knowledge of the gross cellular anatomy of the female pelvis now available, in appraisal of extension of cancer beyond the confines of the uterus, local metastases may be expressed in terms of interstitial involvement, superseding our heretofore hazy understanding of lymphatic drainage.

2. Experience in dissection of the pelvic cellular tissues makes it conclusively evident that radical vaginal hysterectomy, irrespective of the talent of the surgeon, cannot rival the thoroughness of procedure which is possible in extirpation of the cancer-vulnerable tissues from the abdominal approach.

3. Instillation of 20 cubic centimeters of 1: 1,000 methylene blue solution into the empty bladder is adequate protection against vesical injury during difficult pelvic operations.

4. In cases with firm welding of the bladder to the uterus, thus making hysterectomy hazardous, the operative technique is simplified by leaving the bladder undisturbed until the uterus is otherwise completely freed, when separation may be effected without effort, along natural lines of cleavage.

5. In many cases with formidable pelvic pathological lesions the operative hazard may be lessened and the thoroughness of the procedure increased by simple freeing of the uterus on one side, integrated with exposure of the ureter and radical removal on the more involved side, and the procedure perhaps further modified to advantage by removal from side to side rather than by the more usual, established technique.

6. It is helpful to examine patients with cervical cancer under anesthesia—particularly those with a hidden or extensive lesion—in order to determine the extent of the process, to evaluate the intricacies of the therapeutic problem which confront us, and to administer treatment to greatest advantage. Subsequent re-examinations under anesthesia are of invaluable aid in a considerable percentage of cases.

THE RESULTS OF THE TREATMENT OF BILOCULAR AND DIRECT INGUINAL HERNIAS

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THE recent reports of the results obtained in the treatment of primary bilocular or mixed hernias and direct inguinal hernias are quite discouraging. It has been stated that the incidence of recurrence in these types of hernias alone varies between 18 and 32 per cent (1, 3, 7, 13). In this article we are presenting a comparative study made in a majority of primary inguinal herniorrhaphies of the bilocular and direct types performed in our Surgical Service between December, 1934, and April, 1938. During this period primary inguinal hernias totaling 1,161 were corrected. However, in this report we are including only the studies made in 711 of these herniorrhaphies which have been properly followed up. The types of hernias encountered in this follow-up group with very few exceptions were either of the bilocular type or of the direct type. This series in itself is considered unique inasmuch as the group of patients operated upon were men between the fourth and fifth decades of life. So far as we have been able to ascertain, an exclusive report in the surgical literature dealing with the results obtained following hernioplastic procedures in individuals of this age group has not been made. It is a well known fact that between the ages of 40 and 50 years, the tendency for recurrence of hernias is at its highest (1, 7), and in this series 99 per cent of the cases reported are in this age group.

The group of patients operated upon were all ex-service men. The average age was a little above 43 years at the time of operation. As would be expected, a large number of these patients showed early manifestations of degenerative disorders characteristic of this age group such as chronic cough or bronchitis, arteriosclerosis, hypertensive and articular disorders, etc. In general the surgical risk was considered fair in most instances. In the follow-up studies it was possible to contact adequately a total of 537 individuals or patients on whom a total of 711 herniorrhaphies have been performed. Of this total, 322 individuals (426 herniorrhaphies) have been examined by the patients' private physicians, by industrial surgeons at the place of their employment, by us personally, or by other Government physicians. In 215 instances (285 herniorrhaphies) the statements made by the patients have been taken as prima facie evidence of the results obtained following the operations and the reported percentage of recurrences in this group is higher than in the first group (Tables II, III, IV). These statements are considered by us fairly reliable for all purposes as it has been our experience to find that in most instances the patient's complaints and history in regard to hernias of any kind tally with the regional findings on examination. To be consistent in our statistical analysis, we have taken into consideration that "local bulging" in any degree whether reported by examinations of other physicians or by simple correspondence with the patients is evidence of recurrence of the hernioplasty. We all are aware that local bulging *per se* following hernioplastic procedures is not always a manifestation of a recurrence, and that the absence of bulging and local symptoms is not always a sign of satisfactory results following the operation. Nevertheless, we feel that the statistical error if any in our computations as done would be evenly balanced. In the comparative studies of the cases followed up we have concentrated our attention on the following points of importance: (1) types of hernias found as to side and sac formations, (2) comparative values of the technique used, (3) relative value of the different suture materials used, (4) incidence of recurrences, (5) miscellaneous data, infection, mortality, complications, etc.

TABLE I.—TYPES OF HERNIA

	Cases	Cases
Right sided		333
a. Femoral	5	
Left sided		242
a. Femoral	4	
b. Sliding	3	
Bilateral		293
a. Femoral	4	
b. Sliding	1	
Total cases		868
Total hernias		1,161

1. *Types of hernias found.* From the data in Table I we have found that the right inguinal hernias are about 35 per cent more frequent than the left inguinal hernias and only slightly more common than the bilateral inguinal hernias. The percentage of femoral as well as of sliding hernias

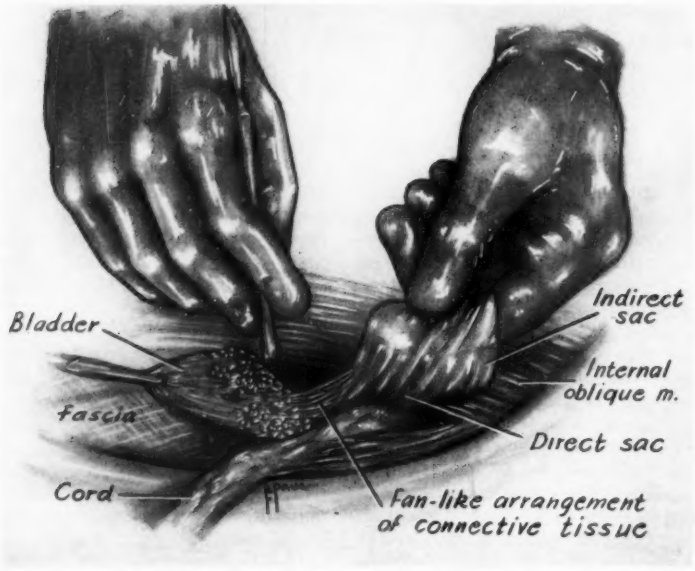

Fig. 1. Illustration demonstrating the final stage in the dissection of the direct locules from the bladder wall. The fan-like arrangement of the intervening areolar tissue serves as excellent line of cleavage. The deep epigastric vessels have been displaced medially and forward. The direct locules have been reduced with the formation of one large locule just lateral to the vessels mentioned.

found is considered within the accepted statistical limits (9).

In this series of cases the majority, approximately 66 per cent, of the hernia defects encountered at the time of the operations were of the bilocular type (mixed type, indirect-direct type), with the direct out-pouching of the bilocular sacs predominating. The pure monolocular direct defects were found in approximately 30 per cent of the cases. The pure indirect defects, femoral hernias, and sliding hernias, were found in approximately 4 per cent of the total patients operated upon. This finding does not conform with the usual conception that the direct monolocular hernias have a much greater incidence after the fourth decade of life than any other types of hernial defects (3, 7). It has been our routine to approach all hernial sacs at the level of the internal rings and invariably we have been able to demonstrate indirect locules or sacs varying in size from a few centimeters in dimension to large hernial sacs. By approaching the hernial sac at the level of the internal ring, it is possible to dissect all the direct locules and at the same time to carry on a digital exploration of the under surface of the inguinal floor. In this exploration the surgeon gains important information such as the strength of the fascia transversalis, the presence or absence of diverticular defects, and the patency of the femoral ring. By this method of approach there is less likelihood of overlooking unusual defects. Many surgeons prefer to invert the direct protrusions without excising the peritoneal locules, whereas they invariably dissect and excise the oblique peritoneal sacs. This is obviously inconsistent and paradoxical (the non-removal of the direct peritoneal locules), and it is our opinion that this is one of the reasons for the high incidence of recurrences following the repair of direct and bilocular inguinal hernial defects. Czerny, quoting Coley, attempted in the later part of last century to correct oblique hernias without excising the indirect sacs and his results were disastrous. On the other hand Bassini's results were relatively brilliant as he stressed and practiced the high ligation and excision of the indirect sacs.

2. *Comparative value of the techniques used.* It is hard to evaluate the merits of a given surgical procedure when this is done by different surgeons. However, we are in a position to do this with the

least possible error as our surgical staff is compact and closely supervised. All the surgeons who performed the herniorrhaphies reported in this series were very meticulous in the excision of all locules of the hernial sacs (Fig. 1) and similarly all carried out the digital exploration of the under surface of the inguinal canals. In the reconstructive procedures some variations were followed and we have been able to classify them accordingly.

a. In 240 herniorrhaphies the reconstruction of the posterior walls was performed according to the method of Bassini with slight variations and the use chiefly of 20 day chromic catgut sutures. In this group there have been reported 31 recurrences, or 12.8 per cent of the total repairs done according to this method.

b. In 457 herniorrhaphies the reconstruction of the walls was carried out according to a method recently described by us (12), in which we used strictly a fascial closure and fine black silk sutures. In this group there has been found 34 recurrences, or 7.5 per cent of the total repairs done according to this method.

c. In 14 herniorrhaphies the reconstruction of the posterior walls was carried out according to the method of Gallie or its modifications and sutures were of fascial strips either from the aponeurosis of the external oblique or from the fascia lata. In this group we have 2 recurrences, or 14.2 per cent of the total repairs done.

As will be noticed, the percentage recurrence regardless of the technique followed is far below the 18 to 32 per cent reported by many authors in their series of bilocular and direct hernias (1, 3, 7, 13). Dr. Gallie, as quoted by Nilsson in the Hunterian Lecture delivered by him at the Royal College of Surgeons in England in 1924, stated that the examinations of hospital records showed a recurrence rate of 40 to 50 per cent of all direct hernias operated upon. Technically our series of cases for comparative purposes falls in the group of direct hernias. We attribute the low percentage recurrence in this series presented as compared with other reported series (1, 3, 7, 13) to two main factors; (1) the thorough excision of all peritoneal locules in the hernial defects; (2) we rarely overlook minor concomitant defects responsible to some extent for recurrences, such as diverticula, large femoral rings, attenuated fascia transversalis or Poupart's ligament, etc. This low recurrence factor is achieved only by the help afforded by the routine digital exploration of the under surface of the inguinal floor which permits the surgeon in the reconstruction to introduce the minor variations needed to correct defects which might be primary factors in causing recurrence.

3. *Relative values of different sutures used.* (a) Silk suture was used exclusively in 457 herniorrhaphies with a percentage recurrence of 7.5 per cent. It was also used as supplementary suture in 14 other cases. In our experience the rate of recurrence when silk is used is much lower than when chromic catgut or fascial strips are used. It has been reported (8) that when silk is used the postoperative exudative phase of the healing process is much shorter than with chromic catgut and also that the fibroblastic processes are of greater magnitude or extent. It is obvious that with silk sutures the final strength of the wound is greater than with catgut. These two virtues in silk make this suture the ideal one for the correction of hernial defects when fibroplasia is of utmost importance (11). Moreover silk is a well standardized suture. One disadvantage of silk is that it is a harder suture to handle and another disadvantage is that the technique has to be observed more closely as any infection means prolonged postoperative drainage from the wound which persists until the infected silk sutures are extruded. Occasionally a second minor operation is required to fetch the infected sutures in order to expedite the healing of the wound.

b. Chromic catgut suture was used exclusively in 240 herniorrhaphies with a percentage recurrence of 12.8 per cent. This suture has its advantages over silk but these are chiefly technical ones. It is an easier suture to work with and as a rule is a well standardized suture. Similarly in unexpected wound infections the drainage is of shorter duration and a second minor operation is rarely indicated. However, its power to stimulate fibroplasia is much lower than that of silk, and therefore it is a less advantageous suture in the repair of hernial defects.

c. Autogenous fascial strips as suture material was used in 14 cases with a percentage recurrence of 14.2 per cent. In our opinion this suture has no special advantages over silk or chromic catgut sutures except in those instances in which there is not only relaxation but also structural weakness of the fascia transversalis. Similarly this suture is of value in all instances in which the fascia transversalis is rather short and its approximation to the under shelf and shelf of Poupart's ligament has to be made under some tension. There have been discouraging reports (1, 3) about the value of fascial strips in the repair of hernias but there is no doubt in our minds that this suture is extremely valuable in the cases indicated.

4. *Incidence of recurrences.* The incidence of recurrences as shown in Tables II, III, and IV seems to be higher in the first 3 years after the

TABLE II.—RIGHT INGUINAL HERNIORRHAPHIES

Follow-up in months	Number of cases	Recurrences
6 to 12	17	5
13 to 24	59	7
25 to 36	54	7
37 to 48	61	6
49 to 54	26	2
Total cases	217	27
Examined by physicians	121	12
Direct correspondence	96	15

TABLE III.—LEFT INGUINAL HERNIORRHAPHIES

Follow-up in months	Number of cases	Recurrences
6 to 12	15	5
13 to 24	37	1
25 to 36	30	2
37 to 48	42	1
49 to 54	22	0
Total cases	146	9
Examined by physicians	97	4
Direct correspondence	49	5

TABLE IV.—BILATERAL INGUINAL HERNIORRHAPHIES

Follow-up in months	Number of cases	Recurrences right	left	bilat.
6 to 12	18	1	1	0
13 to 24	49	0	1	2
25 to 36	42	0	0	5
37 to 48	44	2	0	4
49 to 54	21	0	0	2
Total hernias	348	3	2	26

Examined by physicians, 104 cases, 208 hernias; 12 cases, 20 hernias.
Direct correspondence, 70 cases; 140 hernias; 6 cases, 11 hernias.

TABLE V.—COMPLICATIONS

Pneumonia, all types	3
Accidental opening of bladder	2
Abscess space of Retzius	1
Severe paralytic ileus	1
Volvulus with obstruction	1
Acute hemorrhagic nephritis	1
Infection of wound	13
Orchitis	7
Transitory cystitis	2

operations. Of a total of 67 recurrences, 49 of them, or 71.2 per cent, appeared during the first 3 years. Thereafter the incidence of recurrences seems to taper down. In a recent analysis done by us in recurrent hernias operated upon we found that the recurrences similarly were higher in the first 3 years. It might be interesting to call attention to the fact that right inguinal herniorrhaphies have a much higher tendency to recur than the left inguinal herniorrhaphies. The probable explanation of this phenomenon is a simple one. The stresses to which the right inguinal repairs are exposed are greater and of higher frequency than those acting on the left inguinal repairs—the right-sided musculature is normally more active and stronger than the left-sided musculature of the body. The recurrences in bilateral herniorrhaphies are no more frequent than those of right inguinal herniorrhaphies. This finding does not corroborate the usual conception that the probabilities of recurrences are higher in bilateral herniorrhaphies than in unilateral herniorrhaphies. As a matter of fact there is practically very little difference between the recurrence rate of bilateral herniorrhaphies and right-sided herniorrhaphies in this series presented. The total recurrences in this series are so far 67, or 9.4 per cent, of the total 711 cases followed up. It is obvious that this percentage of recurrences will be higher if all the cases followed up had been followed for a period of 54 months. However, we feel that still the number of recurrences would be comparatively a low one.

5. *Minor data.* a. Anesthesia. In 770 cases out of a total of 868 cases spinal anesthesia was used. Local anesthesia and other anesthetics were used only when there was a definite contra-indication for the use of the spinal anesthesia and also in those few cases in which the spinal anesthetic failed to be fixed partially or entirely. In this series spinal anesthesia in the type of cases in which operation was done was found to be invaluable.

b. Complications. In addition to these major complications there were other minor ones such as postoperative acute urinary retention, mild nausea, mild vomiting, headaches, gas pains, etc.

c. Mortality. Four deaths occurred in the total of 868 patients operated upon. These deaths were attributed to the following complications: volvulus with obstruction, autopsy; severe paralytic ileus, autopsy; acute hemorrhagic nephritis, no autopsy; and postoperative pneumonia, no autopsy. The mortality rate was 0.46 per cent.

d. Postoperative care. In unilateral uncomplicated hernias the patients stayed in bed an average of 8 days. In bilateral uncomplicated hernias the patients were kept in bed on an average of 12 days. Patients were instructed to move from side to side to avoid congestive pulmonary changes. Sedatives and laxatives were given as indicated. At time of discharge all patients were instructed not to do any lifting in excess of 20 pounds for a 6 months' period.

SUMMARY AND CONCLUSIONS

1. An analysis of 711 herniorrhaphies performed in men between the fourth and fifth decades of life has been presented.

2. The dissection and excision of all locules or peritoneal sacs is desirable to reduce the percentage recurrence following the repair of bilocular and direct hernias.

3. Silk is the ideal suture in the repair of hernias and its advantages and disadvantages have been pointed out.

4. The indiscriminate use of fascial strips does not decrease the recurrence percentage as compared with silk or chromic catgut when properly used.

5. In 711 herniorrhaphies followed up there have been so far 67 recurrences, or 9.4 per cent of the total.

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PULMONARY EMBOLISM

A Statistical Review of Cases from 1929 through 1938

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THE drama of medicine and surgery is all too frequently marred by the tragedy of pulmonary embolism. A complication which will enter into or cause the deaths of 3,068,000 (2) of the people now alive in the United States merits the serious consideration of the medical profession as a whole.

This study has been made to determine, if possible, in which patients this complication may be suspected, and also to find the common symptoms, if any, that are present at the time of embolism.

In the period from 1929 to 1938, inclusive, 194 cases of pulmonary embolism occurred or 0.184 per cent of the total admittances (Table I). Of these, 104 followed surgery, or 0.434 per cent of the total operative cases, and 90, or 0.394 per cent of purely medical cases.

One hundred and thirty-three of these cases came to autopsy, that is, pulmonary embolism was found in 2.65 per cent of the deaths and in 4.35 per cent of all cases in which necropsy was done. Barker, in reviewing all the cases of pulmonary embolism at the Mayo Clinic during a 13-year period, found that pulmonary embolism accounted for 5.9 per cent of all postoperative deaths.

Table II shows that in only 20 cases—13 surgical and 7 medical or 15.4 per cent of the patients coming to autopsy—had correct diagnoses been made.

The number of emboli occurring in each patient was determined by reviewing the histories very carefully, particular attention being paid to observations recorded by the nurses. Fifty-four of these patients had shown evidence of previous emboli, which, if recognized, might have responded to some form of treatment and, in a fair percentage, have avoided fatal outcome.

The number of emboli occurring in each patient was 1 in 140 patients, 2 in 26 patients, 3 in 19, 4 in 2, 5 in 1, 6 in 1, 8 in 1, and 4 patients had multiple emboli. In the last 4 patients the emboli were not determinable from history but were found at autopsy. Of the 140, many survived the crisis for hours, and active treatment might have benefited them. In only 45 of the 154 fatal cases did death occur in less than 1 hour. Fifty-five patients lived from 1 to 24 hours and 47 lived more than 1 day (Fig. 1).

The origin of the embolus could be determined either clinically or by autopsy in only 128 cases. Thrombi were found in the femoral veins in 75, with 39 on the right and 36 on the left. Mural thrombi were next in frequency, occurring in 44 cases. The other sites are as follows: iliacs, 27; prostatic plexus, 19; saphenous, 4; varicose veins, 4; site of trauma, 6; pelvic veins, 3; miscellaneous, 3.

The site of lodgment of the embolus was most commonly in the pulmonary branch to the right lower lobe. Figure 2 shows the frequency in all parts of the pulmonary circulation.

ETIOLOGY

The etiology of pulmonary embolism is the etiology of venous thrombosis plus other factors in the individual which may predispose to the fatal outcome. Robertson, in analyzing 146 cases, found an average age of 44 years. There were 82 males and 64 females.

In our series of 194 cases, 96 were males of an average age of 57 years: 98 were females of an average age of 56 years: the average age of the entire group was 56 and a fraction years (Fig. 3). The most common age at which pulmonary embolism occurred was in the decade of 61 to 70 years; 46 per cent of this series occurred between the ages of 51 to 70 (Fig. 4).

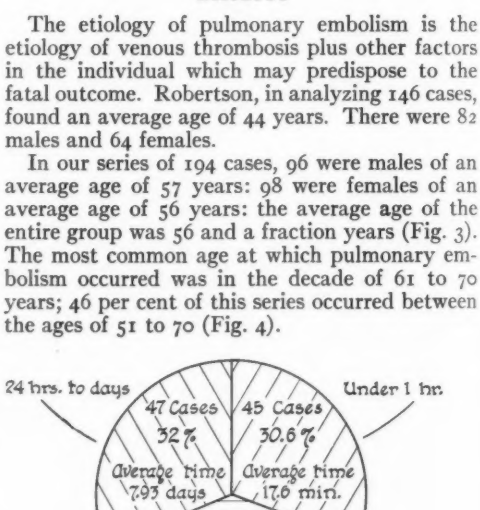

Fig. 1. Time interval between last embolism and death—147 cases.

From St. Luke's and St. Mary's Hospitals.
Presented before the Duluth Surgical Society, March 16, 1939.

TABLE I.—ANALYSIS OF PULMONARY EMBOLISM CASES AT ST. MARY'S AND ST. LUKE'S HOSPITALS

	Cases	Per cent
Total admissions	105,284	
Surgical admissions	23,944	
Medical admissions	82,793	
Cases of pulmonary embolism		
All admissions	194	0.184
Surgical admissions	104	0.434
Medical admissions	90	0.394
Total deaths	5,019	
Total autopsies	3,056	60.8
Total pulmonary embolisms, clinical and autopsy	194	
Total emboli proved by autopsy	133	
Percentage of all deaths		2.65
Percentage of all autopsies		4.35
Proved clinically and autopsy	20	
Proved clinically but not autopsy	61	
Recovered	40	20.6*
Died	154	

*Of 194

TABLE II.—CLINICAL DIAGNOSIS

	Autopsy		Clinical	
	No.	Per cent	No.	Per cent
Surgical	13	26.0	37	74.0
Medical	7	22.6	31	77.3
Total	20		68	

Nutrition. Obesity is claimed by most reviewers to be a predisposing factor in the cause of pulmonary embolism. This, however, did not appear to be true in my series.

NUTRITIONAL STATUS

		Cases	Per cent
Entire group	194		
Above average (obese)		28	14.43
Below average (emaciated)		49	25.25
Average		61	31.44
Not stated		56	28.86
Surgical group	104		
Above average		12	11.5
Below average		21	20.1
Average		36	34.6
Not stated		35	33.6
Medical group	90		
Above average		16	17.7
Below average		28	31.1
Average		25	27.7
Not stated		21	23.3

An interesting observation of this small group is shown in Figure 5, which is interjected for its curious interest only, and most probably has no reason other than coincidence, unless the effects of business may react through the neurovegetative mechanism, thereby increasing the seriousness of what might otherwise have been a minor or overlooked episode (6, 9).

Crawford and Mohler claim that cardiovascular insufficiency favors embolism and is present in 50 to 95 per cent of the necropsies of patients dying as a result of this complication. In the 90 medical cases in my series, 61 per cent showed some form of cardiac pathology.

PATHOLOGICAL LESIONS

	Cases
Cardiovascular	
Cardiac—includes all cases in which cardiac pathology was primary or associated	55*
Coronary sclerosis and occlusion	19
Arteriosclerotic and hypertensive heart disease	18
Valvular	10
Myocardial	10
Thrombophlebitis	10
Decompensation	9
Auricular fibrillation	8
Endocarditis	2
Varicose veins	2
Peripheral vascular disease—arteriosclerosis	1
Cerebral accidents	4
Total	92
Infectious	
Typhoid fever	1
Parotitis	1
Undulant fever	1
Erysipelas	1
Bursitis	1
Total	5
Urologic	
Hypertrophy of prostate with obstruction	3
Kidney infections	3
Chronic nephritis and uremia	2
Cancer of bladder and cystitis	1
Total	9
Metabolic	
Thyrotoxicosis	2
Diabetes mellitus	1
Total	3
Gastro-intestinal	
Hemorrhage	3
Carcinoma of pancreas	1
Carcinoma of bowel	1
Perforations of hollow viscus	1
Total	6
Miscellaneous	
Idiopathic epilepsy	1
Blood dyscrasias	2
Pelvic infection	1
Total	4

*61 per cent of all medical cases.

The type of operation no doubt is a big causative factor in emboli following surgery. Time

consuming and shocking operations, through their physiological upset of the blood constituents, may contribute to thrombosis and ultimately to embolus. Major surgery is, however, not the only factor, as in this series embolism followed a cataract operation, phrenic exeresis, and the removal of a needle from the thoracic wall. Of the 104 surgical cases, 68 occurred after abdominal operations, 30 of which were in the upper abdomen.

SURGICAL OPERATIONS

	Cases
Head and neck	
Subtotal thyroidectomy	3
Phrenic exeresis and scalenotomy	1
Bilateral cataract	1
Total	5
Thorax	
Radical amputation of breast	4
Thoracoplasty	1
Removal of needle from thoracic wall	1
Pericardectomy	1
Total	7
Upper abdomen	
Gall bladder and bile ducts	17
Gastric operations—malignant, 6; benign, 4	10
Exploratory (inoperable malignancy)	3
Total	30
Lower abdomen	
Appendectomy without drainage	7
Colostomy (malignancy)	3
Hernias—inguinal, 3; femoral, 1; umbilical, 1; incisional, 1	6
Appendectomy with drainage	2
Release adhesions	2
Resection of malignancies of large bowel	2
Total	22
Gynecologic	
Oophorectomy, salpingectomy, or both	4
Supravaginal hysterectomy	3
Panhysterectomy—malignant, 1; benign, 4	5
Radium insertion (malignancy)	2
Septic abortion	1
Retained placenta	1
Total	16
Urologic	
Transurethral prostatic resections	3
Suprapubic prostatectomy	2
Cystostomy	2
Nephrectomy	1
Aspiration of hydrocele	1
Total	9
Extremities	
Ligation or injection of varicose veins	2
Amputation of leg	1
Removal of semilunar cartilage	1
Arthrotomy	1
Total	5
Fractures*	
Neck of femur	4
Compression of vertebræ	3
Ribs	2
Knee	2
Ankle	1
Os calcis	1
Pelvis	1
Pathological, femur (sarcoma)	1
Shaft of femur	1
Skull	1
Shaft of tibia	1
Total	18

*3 were multiple.

DIAGNOSIS

The diagnosis of a massive, sudden pulmonary embolism, which usually results fatally, is not difficult. The diagnosis of the minor showers or single emboli challenges the clinician, if anything is to be done to prevent the later larger and fatal emboli.

Various recurring painful areas in the chest, unexplained low grade fevers for which the condition of the wound will not account, pain about the operative site out of line with the findings, and finally a nurse's note that the patient is "very apprehensive," have all been noted as prodromal symptoms. These complaints should place one on guard that he is dealing with a potential serious embolus. Robertson found an unexplained low grade fever in 18 per cent of his patients while in this series this occurred in only 7 per cent.

FEVER INCIDENCE

	Cases	Per cent
Normal temperature curves	29	15
Low grade fevers	14	7
Septic temperature	22	11.3
Rise at or shortly after onset of embolus	119	61.0
Average degree of temperature rise at or after onset of embolus		1.95

In reviewing the charts in search of the symptoms to explain the time of lodgment of the embolus, it was noted that accompanying the increased pulse rate there was a rise in temperature. This occurred in 119, or 61 per cent of the series. There was an average rise in temperature at or immediately following the symptoms of dyspnea, cyanosis, pain, etc., of 1.95 degrees. Usually these reactions subsided in 8 to 24 hours unless other complications ensued.

With the lodgment of an embolus of consequence, symptoms of disaster are present. These usually are manifested by fainting, weakness, cyanosis, dyspnea, pain in the chest, hemoptysis,

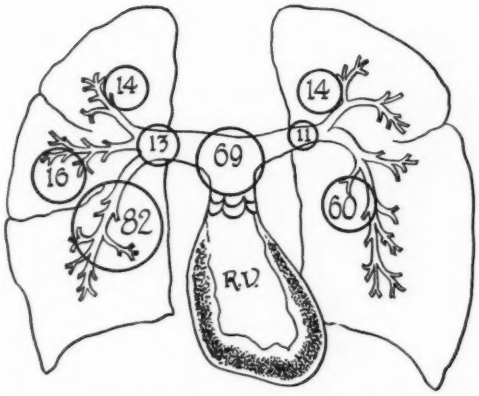

Fig. 2. Site of embolus, determined clinically and by autopsy.

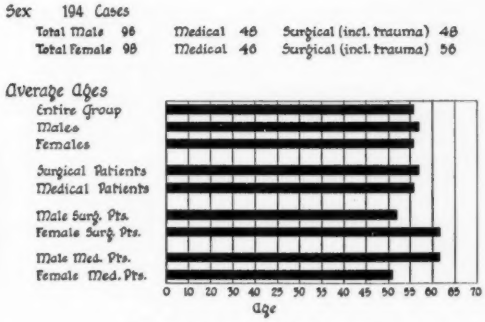

Fig. 3. Sex and age incidence.

or only grave fear of impending danger. These are usually accompanied by a rapid thready pulse, frequently cardiac arrythmias, and extreme prostration. A fall in blood pressure is practically always present.

Immediate physical findings other than those already mentioned are nil. According to Robertson, if a main pulmonary branch is occluded, findings of pulmonary congestion soon develop in the opposite lung, due to the increased blood flow. If only one lobar branch is occluded, findings of congestion will be present in the other lobe or lobes of the corresponding lung. If there is a massive block of the main stem, immediate death ensues due to cerebral anemia. This is accompanied by collapse, syncope, pallor, and sudden death.

With the onset of pain, pleurisy may be complained of and diagnosed as such by the friction rub to be heard. Probably there will also be limited expansion on the affected side. Bloody sputum may occur early but is usually a later finding.

In the event a marked obstruction to the pulmonary circulation has occurred, evidences of acute dilatation of the right ventricle and pulmonary conus may be present, as stated by White. Peripheral venous dilatation, sometimes pulsation, may be noted. This is usually associated with marked accentuation of the second pulmonic sound due to the increased pulmonic pressure. Increased pulsations may be noted in the left second and third interspaces as well as a loud systolic murmur to be heard in this area.

Pulmonary infarction with its findings of complete or incomplete consolidation will occur later, only if there be some impedance to the return venous flow of blood (4).

In patients who have exhibited minor symptoms of emboli, search should be carefully made for the slightest evidence of thromboses. O'Ware (8) has found characteristic tenderness of the sole of the foot accompanying thrombosis of the foot or leg. Abdominal, prostatic, and uterine veins produce few or no symptoms but psychic apprehension; vague indefinable discomforts in the abdomen or legs frequently precede the occurrence of embolism. Robertson advises that in these patients a definite effort be made to determine whether or not they are "thrombophiles." He states that if the clotting time of the blood is shortened after the fourth postoperative day or at any time during convalescence, it is suggestive of thrombosis. The platelet count may be of value in determining a "thrombophile" if done by one experienced in this work. He states that if the platelet count falls below normal and continues low, a thrombus may be developing. Lastly, an accelerated sedimentation rate after the normal postoperative expectancy (young patients, 8 to 10 days; older, 10 to 20 days), should be regarded with apprehension.

Roentgen-ray diagnosis of pulmonary embolism has been not only unsatisfactory but detrimental to the patients in this group. Roentgen-ray diagnoses had been made on 51 of these patients who later came to autopsy. Diagnosis of pulmonary embolism questioned was made on 1 patient.

Roentgen-ray diagnoses in 51 autopsy proved emboli were as follows: passive congestion, 10; negative, 9; slight bronchopneumonia, 7; suspected pneumonia or bronchitis, 6; increased bronchovascular markings, 4; thickened pleura or fluid, 4; atelectasis, 2; consolidation, 2; haziness, 2; miscellaneous—singulars, 4; pulmonary embolism (?), 1.

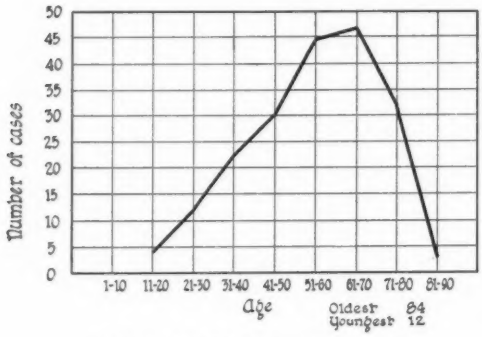

Fig. 4. Age incidence—194 cases.

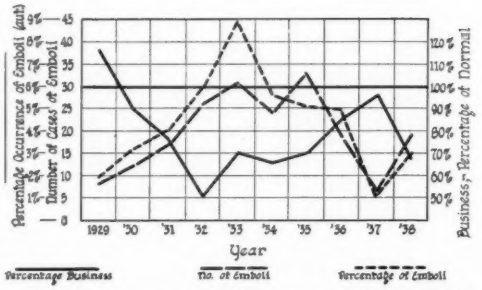

Fig. 5. Occurrence of pulmonary embolism in relation to business activity.

Differential diagnosis. The condition most commonly confused with pulmonary embolism is acute coronary occlusion. A mistaken diagnosis in these conditions will not, as a rule, do any harm to the patient because the treatment for one is good treatment for the other and logical treatment for either can in no way harm the other. In coronary occlusion the pain is usually much more severe than in embolism and is more frequently referred to the central regions. The pain of embolus, when present, is usually around the costal margins or laterally in the chest. It is usually aggravated by respiration while that of coronary disease is not affected. Dyspnea and cyanosis are usually more marked in coronary occlusion. The other symptoms of fainting, shock, weakness, sweating, collapse, vomiting, etc., are common to both conditions. The history of anginal attacks, dyspnea or other symptoms of cardiac disease may assist. White, McGinn, and White and corroborated by Barnes have shown that distinctive characteristic electrocardiographic changes take place in both conditions, and when there is a question as to diagnosis, this procedure should be availed of. Barker states that he believes that the electrocardiogram is the most valuable diagnostic procedure when a pulmonary embolus is present.

Spontaneous pneumothorax should be readily differentiated by the rather marked physical findings in the chest, shifting mediastinum, and finally a roentgen-ray examination of the chest, if deemed absolutely necessary, should clinch the diagnosis.

Atelectasis usually occurs sooner after periods of unconsciousness and though the symptoms are frequently the same as in embolism, the severe early cough, the lack of pleural pain, the absence of pulmonary artery back pressure, and finally the roentgen-ray may all assist in making the diagnosis.

The diagnosis of postoperative or hypostatic pneumonia should never be made until pulmonary embolism is ruled out. In my opinion, many of the so called postoperative pneumonias are primarily emboli later developing pneumonic findings. The treatment of pulmonary embolism will in no way interfere with what pneumonia should have, and if a frank pneumonia is developing, the diagnosis should be clear after 24 hours has elapsed and no time will have been lost to the most hazardous embolism.

Pleurisy, with its usual pain, friction rub, mild fever, etc., should always be regarded as evidence of embolism in older postoperative patients or in medical cases presenting symptoms of impaired circulation.

TREATMENT

The treatment of pulmonary embolism has been very well covered in the literature and hence will not be reviewed in this article. A brief summary of important points, however, might not be amiss.

The preventive treatment should be directed toward the prevention of stagnation to the return flow of blood. This can be accomplished by active and passive exercises, hyperventilation, maintenance of blood pressure level, and preventive or active treatment of ileus. Dietary influences on the coagulation of blood may enter into the predisposition to thrombosis and ultimately emboli. Kugelmann, Mills, and Clark (8) have all shown that the low fat, low protein diet is the least conducive to thrombotic changes, and recently gelatine has been found to lower rather markedly the bleeding and coagulation time of patients submitted to tonsillectomy (7). It has been stated by some authorities (1) that when heparin is used by continuous intravenous drip, thrombosis will not occur.

Active treatment will depend upon the recognition of the minor symptoms of small emboli, thereby being prepared to meet the major crisis. Oxygen should be immediately available. Sterile syringe, needle, and tourniquet together with a solution containing ½ grain of papaverine hydrochloride should be in the room for immediate intravenous administration (3, 5, 6). This should be followed by injections of fairly large doses of morphine and atropine. The patient should be in unceasing attendance by an intelligent nurse. On this regimen one of our patients recently recovered from what appeared to be a large embolus.

SUMMARY

A statistical review of the cases of pulmonary embolism occurring in 105,284 hospital admittances has been given.

Etiological and pathological findings as they may enter into pulmonary embolism have been given from a statistical standpoint.

Accompanying the usual symptoms of pulmonary embolism an average rise of temperature of 1.95 degrees was found in 61 per cent of these patients.

X-ray diagnosis of pulmonary embolism is not only unsatisfactory but detrimental to the patient's welfare.

Electrocardiographic tracings are a valuable procedure in the diagnosis of pulmonary embolism.

Careful study and observation may lead us to the determination of the type of patient predisposed to pulmonary embolism, and it is to this end our efforts in the future should be directed.

Prophylactic treatment of active exercises, massage, prevention of acidosis, the use of heparin, psychic relaxation, and dietary control may avoid emboli.

Active treatment with antispasmodics and sedatives offers some hope after the crisis has occurred.

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VITALLIUM NAILS IN FRACTURES OF THE HIP

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THE solution of the "unsolved fracture" is gradually unfolding and the essential features of its treatment are agreed upon with the exception of the type of metal device used for fixation. Whitman's method of reduction, described in 1904, has become universally recognized as a standard procedure. The fixation of hip fractures with metal appliances has been well worked out by Smith-Petersen, Moore, Martin, and many others. The technique of exposing the fractures and inserting the fixation devices is becoming more simplified and less subject to error. The criteria of proper postoperative care and protection of the fractured extremities are coming to be agreed upon by most surgeons. In short, most of the steps in treatment of fractures of the hip have become well enough established that there is little disagreement about them.

One remaining source of argument in this matter concerns the choice of metal alloys of which the various nails are made, and it is this which we hope to clarify in our present paper. For 3 years we have been experimenting and reporting our findings on the reaction of bone to metals (24 to 28). Over and over again we have demonstrated that most metals commonly used in bone surgery produce destructive electrolytic effects which cause the appliances to loosen. There is yet much confusion over the causes of failure when metal nails in the hip become loose and ineffective. For instance, many surgeons have observed that collections of fluid accumulate about the ordinary steel nails, the surrounding bone becomes eroded, and the nails become loose and quite insecure in a comparatively short time (Conwell and Sherrill; Henderson, 10–12; Watson-Jones; Campbell, 4; Gaenslen; MacAusland; Bosworth). This is usually blamed on infection, pressure necrosis, foreign body reaction, or faulty technique which throws undue strain on the bone.

It is our observation that nails become loose (in the absence of infection or technical faults) because of electrolytic activity between the constituent metals in the alloy which is sufficiently intense to destroy adjacent bone. The amount of erosion we have found depends upon the constituents of the alloy and their resistance or relative passivity to electrolytic activity in the body fluids. Nickel or chromium-plated steel nails cause marked destruction of bone because of the galvanic cell action of the dissimilar metals. A single unplated nail of so called stainless steel is composed of many metals, such as iron, chromium, nickel, etc., and the electro-activity between them, which can be demonstrated with a microammeter, will cause bone to be corroded about the nail. Fluid accumulation which seems to indicate infection is more often a body reaction against the local irritation. Cultures of such fluid are invariably negative but chemical examinations usually reveal evidence of metals in solution.

A number of men have removed nails from the hip and noticed that they were eroded and discolored while the adjacent bone was stained with deposits of metal pigments (Campbell, 4; Harris; Speed; Kulowski; Raagaard). Such extensive destruction of metal was ascribed to chemical corrosion or to defective preparation of the nails. It is a striking proof of the intensity of electrolytic activity that it can not only erode bone but cause a metal nail to disintegrate in the body fluids (Figs. 1, 2, 3). In our experiments, chemical analyses of bone specimens from the neighborhood of such metal appliances revealed the presence of particles of the constituent metals of the alloy. Moreover, examinations of the livers and kidneys of experimental animals were also positive for evidence of deposited metals.

It has been pointed out by Harris and Jones and Lieberman that the terms "rustless steel" and "stainless steel" are used to describe many ferrous alloys of widely dissimilar composition. They state further that most surgeons are not qualified to determine the suitability of a proposed alloy for use in the body and are therefore at the mercy of instrument salesmen or must depend on the recommendations of surgical catalogs.

Alloys of various composition have been recommended as suitable for bone surgery and their proponents have stated that they caused no erosion of bone; but tests for electrolytic activity have seldom been performed. Stainless steel (Moore), monel metal (Henderson), chrome steel (Carothers), chrome-nickel steel (Plummer), low tempered tool steel (Lippman), and many more have been utilized in the body with varying degrees of success. We have found that many of these alloys were sufficiently electro-active to pro-

duce a marked current when connected with a microammeter. Also when some of the metals were placed in physiological saline for a short time, chemical examination of the fluid revealed evidence of constituent metals in solution. Consequently, we hesitate to recommend an alloy for use in the body when such proofs of electrolytic activity are demonstrable. For this reason we introduced the non-electrolytic alloy vitallium into bone surgery since it was the only one which quickly became permanently passive in the body.

Raagaard, of Denmark, and Harris, of Toronto, were of the opinion that corrosion of nails and surrounding bone was directly affected by the finish of the nails. They inferred this by pointing out that the more rapid disintegration of metals took place in screw threads or near the ends of the nails. We have likewise observed this but we have seen that corrosion will not take place at any point in a piece of non-electrolytic alloy. *In other words when an alloy is non-electrolytic there are no anodic or cathodic points where currents are initiated.* Our views of this question can be better explained by a brief description of the phenomena of electrolysis about metal appliances.

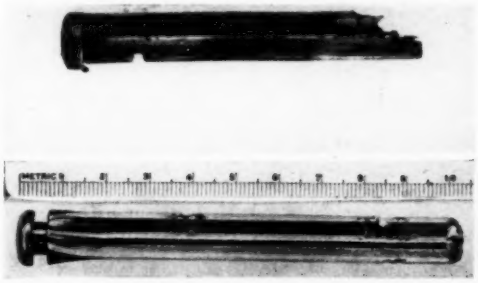

Fig. 1, top. Photograph of steel nail removed from patient by Kellogg Speed. There had been pain, erosion of bone, and absence of union. Note electrolytic disintegration of metal which was sufficient to destroy bone about the nail.

Fig. 2. Photograph of nail of high chromium, high nickel stainless steel, removed by R. I. Harris. Electrolytic activity which corroded the nail caused marked destruction of bone and formation of "sterile abscess" in the wound.

HOW ELECTROLYSIS TAKES PLACE

Corrosion of metal is preceded by electrolysis and electrolysis can take place only in the presence of *moisture.* It is to prevent this that exposed metal is painted. Oxidation furthermore can take place only after electrolysis and corrosion have begun. For instance, Masmonteil points out that the hulls of hydroplanes, although made of corrosion-resistant alloys which would not tarnish, were subject to electrolytic reaction when in an electrolyte like sea water. Also it is a familiar observation of those living near the sea that salt spray causes rapid disintegration of exposed metals. Dissimilar metallic ions must be in close proximity for electrolysis to take place. This does not necessarily mean that two separate pieces of metal must be in contact because the presence of dissimilar metallic ions in an alloy will be sufficient stimulus to set up local currents. Of course, an appliance made of a single chemically pure metal would not be subject to electrolysis when immersed in saline but metals free of impurities are quite expensive and far too soft to be made into screws or nails. In the common alloys, ions of the different constituent metals are scattered about helter skelter so that each mass of particles forms a tiny battery with the adjacent dissimilar particles and gradually produces irregular destruction of the surface comparable to "pit corrosion."

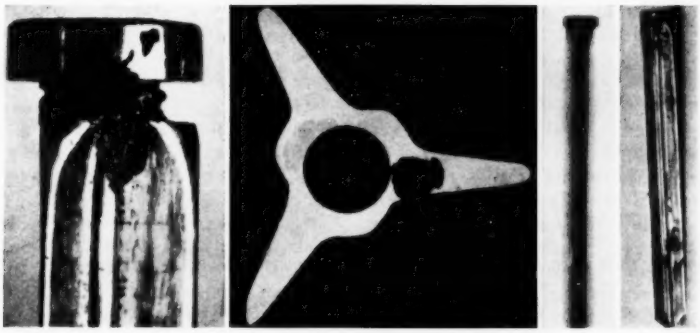

Fig. 3. Photograph, roentgenogram, and cross section of rustless steel nail removed by Otto Raagaard. Electrolytic destruction of metal and bone caused nail to loosen sufficiently to prevent union of fracture.

Fig. 4.

Fig. 4. Mrs. D. N. C. Fracture of left hip anchored with vitallium screw. Roentgenogram taken 3 months later shows solid bony union with no evidence of reaction about the screw.

Fig. 5.

Fig. 5. Mrs. G. H. Fracture of right hip fixed with vitallium Smith-Petersen nail. Roentgenogram 10 months later shows secure attachment of nail to bone with no erosion about it. Fracture solidly healed.

Fig. 6.

Fig. 6. Mrs. H. J., aged 62 years. Fracture of right hip which was treated with vitallium Smith-Petersen nail. Roentgenogram 5 months later shows solid bony union of the fracture with no changes about the nail.

Fig. 7.

Fig. 7. Miss E. F., aged 67 years. Fracture of right hip secured with vitallium Smith-Petersen nail. Roentgenogram which was taken 4 months later reveals good union of the fracture.

Electrolysis of metals is accelerated if the molecular arrangement of the alloy is altered by certain physical forces. Thus if a plain carpenter's nail is bent and placed beside a similar straight nail in saline, the bent nail will corrode more rapidly and break at the point of bending where there is produced continual anodic-cathodic action. A piece of an electrolytic alloy of rough finish or a piece with many irregular surfaces and corners will corrode more rapidly in an electrolyte than will similar articles of the same alloy which are highly polished.

Corrosion, rusting, oxidation, and the more familiar end-stages of the disintegration process are preceded by electrolytic activity between the constituent ions of an alloy. Consequently, metals used in the body, where they are bathed in a salt solution 24 hours a day, must be non-electrolytic if they are to resist corrosion.

HOW ELECTROLYSIS CAN BE PREVENTED

Since metal appliances used in the body cannot be kept dry and since they cannot be constructed of a single pure metal, we have sought an alloy that would be sufficiently strong and yet free of any electrolytic activity in body fluids. Recently it has been brought to our attention that Masmonteil, of Saint-Ouen, had arrived at these same conclusions and had stated that metals suitable for bone surgery must be biologically inert, i.e., free of organisms; mechanically inert, i.e., placed in a proper position on the bone; chemically inert, containing no injurious substances; and physically inert, or free of electrolytic activity. Of the various alloys he tested none satisfied all the postulates. The solution, concluded Masmonteil, "C'est le secret de demain."

Berti-Riboli, of Genoa, has likewise found that metals which erode bone in experimental animals are the ones with the most electromotive force. On the other hand, the metals with least electromotive force, such as 20 carat gold, produced less toxic effect on the bone than any others.

We have found that alloys which produce appreciable current on the microammeter will cause erosion of bone in experimental animals. Conversely, an alloy like vitallium which produces little or no current on the microammeter can be placed in bone without danger of the pathological reactions which Masmonteil has called "electrolytic osteitis."

VITALLIUM ALLOY

In 1936 we reported animal experiments which showed that electrolysis was the important factor in the use of metals in bone and that the alloy vitallium seemed totally inert in human bone (25). Since that time appliances made of vitallium have been used in various ways by many surgeons, including Smith-Petersen, Campbell(5), Badgley, and others, with no case of erosion of bone about the metal appliances.

In the 20 patients in whom we placed vitallium nails in the hip more than 6 months ago, there has not been any evidence of loosening of the nails or erosion of bone. Eighteen of these patients

Fig. 8a.

Fig. 8b.

Fig. 8c.

Fig. 9.

Fig. 8. a, Mrs. W. D. B., aged 60 years. Fracture of right hip treated with vitallium Smith-Petersen nail. Nail was too short and did not secure good hold on capital fragment; b, 3 months later, adduction deformity and displacement of head of femur; c, 8 months later, after nail had been re-inserted in head. Solid bony union, no reaction about nail.

Fig. 9. Miss A. B., aged 76 years. Fracture of left hip treated with vitallium Smith-Petersen nail. Roentgenogram 1 year later shows firm union with no evidence of erosion about the nail.

achieved solid rapid bony union and no interference with healing of the bone. In 1 patient the nail pulled out of the head of the femur because it was not placed sufficiently deep. Even so, it was re-inserted, the fracture healed, and the nail is still tightly implanted in the bone (Fig. 8). The one failure was a similar case in which the nail engaged the head insufficiently and the fracture pulled apart several months after the operation as the patient was getting in a car. This nail was removed and the patient developed a complete non-union of the fracture.

In none of the cases did the wound fail to heal promptly and no fluid accumulated in any hip. Of the 20 patients, 19 attained bony union and the 1 failure in this series was due to a technical fault in the insertion of the nail. Over a period of many months no late complications have developed in any case due to the presence of the metal nail (Figs. 4 to 9).

Vitallium is an alloy of cobalt, 65 per cent, chromium, 30 per cent, and molybdenum, 5 per cent, which seems to be completely passive in the tissues. It is hard, non-malleable and at first was not strong enough. However, we believe that this material has now been toughened sufficiently to be comparable in strength to any of the steels. Nevertheless we know of no instance in which a vitallium hip nail has cracked or broken in the hip. A single vitallium nail will support 500 pounds in a power tester without bending. Appliances made of this alloy must be cast, since it cannot be machined.

On the favorable side, we find that vitallium produces no appreciable current on the microammeter, it is not affected by submersion in saline, and it is electrically passive in the body fluids. Appliances made of vitallium can be used in the body with perfect safety and can remain in the bone indefinitely without the usual haunting fears of the occurrence of late complications. Vitallium screws and nails do not loosen in bone, no fluid forms around them, and normal body healing processes are in no way delayed by the presence of the metal.

Undoubtedly, other equally inert metals will be developed which may be more adaptable to bone surgery. We hope so, and we will pursue our experiments in order to test any new alloy which comes to our attention. Our conviction is that the ideal metal developed by the orthopedic surgeons of tomorrow must meet our qualifications of complete absence of electrolysis in body fluids, negative readings by microammeter test, and absolute inertness in experimental animals or in experimental batteries. Certainly any appliance as large as a Smith-Petersen nail which gains its advantage by surface hold on the bone must be constructed of a non-electrolytic alloy. Vitallium nails meet these requirements more perfectly than any we have encountered in our experimental work thus far.

Other tests are under way because one of us (C.S.V.) is working with the Fracture Committee of the American College of Surgeons in a search for the most suitable alloy for use in bone surgery. Whatever is the final choice, we think we have proved that no metal will remain inert in bone unless it is at first and continuously thereafter electrically passive.

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THE RESULTS OF SURGICAL TREATMENT OF RECURRENT INGUINAL HERNIA

CLAUDE C. BURTON, M.D., F.A.C.S., and RAOUL L. RAMOS, M.D.,
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NUMEROUS operative procedures have been devised for the correction of inguinal hernias, and in their evolution much progress has been made. However, we still have no right to assume a too complacent attitude since the number of recurrences following primary repair, as reported by different authorities (4, 5, 8, 13) upon careful follow-up, varies from 5 to 20 per cent for indirect hernias and 20 to 30 per cent for direct hernias. This is too high. Yet it should not be a deterrent; on the contrary the recognition of this fact should serve as a stimulus to examine meticulously the various factors in the techniques used in an effort to overcome, replace, or reinforce the structural weaknesses that might exist.

This paper is based upon a study of 130 recurrent hernias in 115 patients who were operated upon between January, 1935, and April, 1938. In this series, 109 were primary recurrences, 11 secondary, and 10 tertiary recurrences. The kind of defect in the wall, the technique and type of sutures used, the percentage of follow-up cases at yearly intervals, and the number of recurrences following our operations are discussed and tabulated. From an analysis of these data the main causes of failure of the original repair of the recurrences were as follows: (1) incomplete removal of all locules of the sac; (2) overlooked diverticula; (3) failure to recognize weaknesses of the wall; (4) unphysiological repair of the wall.

1. *Incomplete removal of all locules of the sac.* During one's didactic and early surgical training, too much emphasis is placed upon the textbook classification of hernias; i.e., indirect, direct, and femoral, which in reality represents the end stages of the hernias. Actually many hernias cannot be so classified and any attempt in this direction may cause confusion. However, when the sac is considered in its incipiency as an offshoot of peritoneum which by increased intra-abdominal tension pushes its way through a weakness or defect in the muscular and the all important posterior fascial wall (internal ring, inguinal triangle, and femoral ring), the sequential development of one or more locules of the sac through the concomitant weak points is easily understood. This consideration simplifies and makes much easier the correlation of the findings at operation. Moreover, by systematic approach to the peritoneum at the internal ring where there is almost invariably some bulging, there is less likelihood of becoming confused by the distorted structures. When the peritoneum is opened and the index finger is introduced one is able to diagnose accurately the number of locules, their size, location and relation, and to evaluate the extent of the fascial and muscular weakness.

It has been our contention that the locules of the sac play a more important rôle in the etiology of recurrent hernias than is generally accorded them. In this series of 130 recurrent hernias the types of sacs found are shown in Table I.

These statistics therefore bear out the consensus, which is supported by many published reports, that most recurrent hernias are direct. In recurrent hernias the ratio of direct to indirect sacs is 8:1, whereas in primary hernias the ratio of direct to indirect sacs is 1:5. The chief reason for the former is that the direct portion of the sac was not recognized and not removed at the initial operation. The bilocular sac, which consists of both direct and indirect locules, appears with increasing frequency in patients past middle age, and is concomitant with a dilated internal ring, diminished muscle tone, and insufficiency of the fascia transversalis. This type is more prone to occur in hernias of long standing. Much to our surprise the diverticular type had an incidence of 13.8 per cent and since it is so easily corrected, one can reasonably assume that its presence was not suspected at the time of the original repair. Therefore, we feel that this type of diverticular

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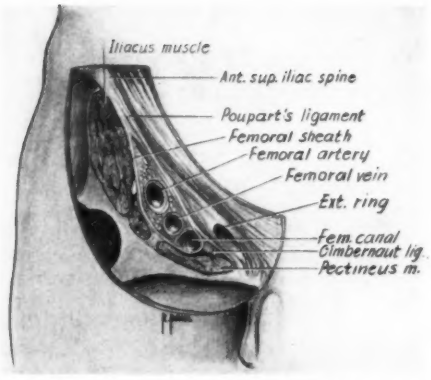

Fig. 1. Sagittal section slightly distal to Poupart's ligament demonstrating the anatomical relations of the principal structures.

defect is not receiving the importance it should as an etiological factor in the development of recurrent hernias.

If routine digital exploration of every sac is practiced to discover possible peritoneal offshoots, this type of recurrence could be reduced. While quite rare, a trilocular sac, indirect-direct-femoral, occurred once in this series and in this case there was marked generalized diminished muscular tone and a dilated femoral ring. Hernia adiposa or fatty masses were noted twice and these could not be differentiated from true herniations prior to exploration. Sliding hernia which was not accurately diagnosed before operation was present once.

2. *Overlooked diverticula.* Andrews and Bissell (4) in 1934 directed attention to diverticula and the part they played in direct hernias. They pointed out diagnostic maneuvers for their detection and introduced plastic corrective measures. The incidence of diverticula in direct primary hernias is reported as less than 10 per cent, while the incidence in our series of recurrent hernias was 13.8 per cent. These diverticula were exclusively responsible for the clinical manifestations of the hernias. Of greater importance, every recurrent diverticular hernia should be considered a diagnostic and not an operative failure as the elimination of the sac and correction of the defect is such a simple, yet secure, procedure that a recurrence is hardly likely. In reviewing our records it has not been clear at times whether the outpouching was a part of the direct sac or a diverticulum which communicated through a stalk. This confusion, however, did not obtain in the other type of diverticulum in which there was a distinctly isolated non-communicating, endothelium lined locule. If the diverticula were limited to the latter, it would be conducive to more accurate statistics regarding this type of hernia.

3. *Failure to recognize weaknesses of the wall.* The cumulative evidence of a decade or more supports the indictment that too often there is a failure to recognize weakness or absence of the transversalis fascia.

TABLE II.—MURAL WEAKNESSES

	Number	Per cent
Relaxation of fascia transversalis	77	59.2
Attenuation of fascia transversalis	36	27.6
Absence of fascia transversalis	8	6.1
Absence of Poupart's ligament	4	3.0
Large femoral ring	3	2.3
No weakness found, fatty masses	2	1.8

In Table II is shown the classification of various mural weaknesses found in this series of recurrent hernias. By relaxation of the fascia transversalis is meant slight bulging of the entire floor due primarily to diminished tone, in contradistinction to attenuation of the fascia in which there is definite thinning of this structure. It is conceded that there may arise at times differences of opinion in interpreting these two types of weakness. However, this distinction is important because it calls for technical variations in the repair of the floor of the canal.

In those cases in which the fascia transversalis is missing, it is unmistakable as there is no resistance offered to the bulging peritoneum. Usually these hernias are huge and frequently the patient has undergone many unsuccessful operations. It is in this type of weakness that a radical hernioplasty is indicated.

A frayed Poupart's ligament is usually associated with absence of fascia transversalis but this is by no means constant. If this ligament is so velamentous, so friable, and its shelf so narrowed that it no longer offers substantial anchorage, then Cooper's ligament (Fig. 2) should be exposed and used in lieu of it.

A large femoral ring should be recognized and if not obliterated, it is a potential source for a recurrence particularly when the peritoneum is attenuated. Therefore, we feel that the type of mural weakness is a better criterion than the sac in determining the most suitable operative procedure.

4. *Unphysiological repair of the wall.* By unphysiological repair of the wall is meant an attempt to correct the defect in the all important fascial floor by utilizing tissues which are not histologically or physiologically related. The

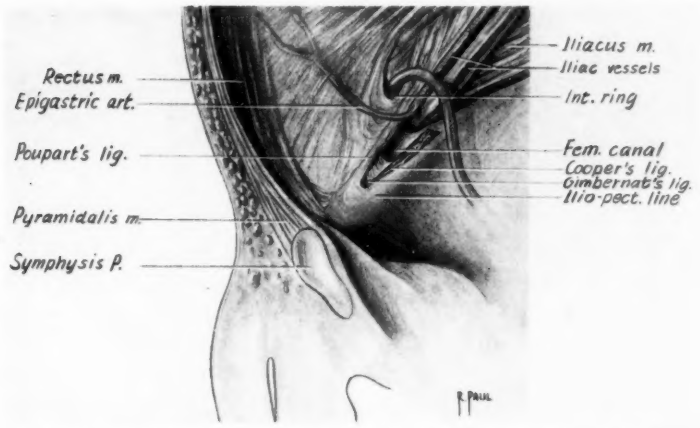

Fig. 2. Posterior inner view of the abdominal cavity minus the parietal peritoneum showing the anatomical relations of the principal structures covered by the inguinal canal floor. Notice the relations of Cooper's ligament to the femoral ring and Gimbernat's ligament.

experimental and clinical work of Andrews, Koontz, Gallie, LeMesurier, Seelig, Turner, Wangensteen, and others should convince the most skeptical that strong fusion takes place only when white fibrous tissues are apposed and that muscle tissue as an auxiliary reparative measure produces no lasting adherence and is worthless in so far as contribution to the formation of a strong supportive floor is concerned. The most frequent error of this nature is the suturing of the internal oblique muscle to Poupart's ligament without regard to the observations of Andrews and Bissell (4), which recently have been corroborated by Anson and McVay, that this muscle is neither constant in its insertion nor in its muscular and aponeurotic components. Therefore, if this muscle is used as a lamina in the repair of the hernia, the resultant union to the ligament will not be strong.

When the insertion of this muscle in the rectus sheath is some distance from the pubic spine the joint actions of these muscles tend to tear away the oblique muscle attached to Poupart's ligament. This simply would leave the wall structurally as it previously existed except perhaps for the removal of the sac. With the realization of this anatomical and physiological principle there has quite naturally followed a retinue of operations the object of which is to accomplish an exclusive fascial closure. When there is a simple dome-like laxity of the fascia transversalis without any attenuation with associated enlarged atonic internal ring, the defect can be corrected by attaching the fascia transversalis to Poupart's ligament. However, if this produces undue tautness or if there is marked attenuation of this fascia, then it becomes necessary to resort to fascial sutures. In the event Poupart's ligament is lacking or is friable, which is more likely to occur after repeated operations, it is suggested that the fascial lamina be attached to Cooper's ligament (6). If there is absence or retraction of the transversalis fascia so that it cannot be made to span the defect in the floor, a pedicle fascial graft may be brought up through the femoral canal, fanned out, and sutured to the lateral margins of the sheath of the rectus, thus replacing the transversalis fascia and making a deeply placed mural lamina. This technique will be discussed later in this article.

OPERATIVE PROCEDURES USED AND INDICATIONS

1. *Routine repair* (11). The method described by us in a previous communication consists in attaching the cleaned fascia transversalis to the shelving border of the inguinal ligament with imbrication of the aponeurotic flaps beneath the transposed cord. A strong fascia transversalis and inguinal ligament must obtain for success of this method. The sac in this technique as well as in the other techniques is dealt with by the excision of all locules and the use of a purse-string suture in the stump. The results following this technique have been very satisfactory. There have been 7.5 per cent recurrences in 457 primary hernias and 14 per cent recurrences in 64 reoperated upon recurrent hernias.

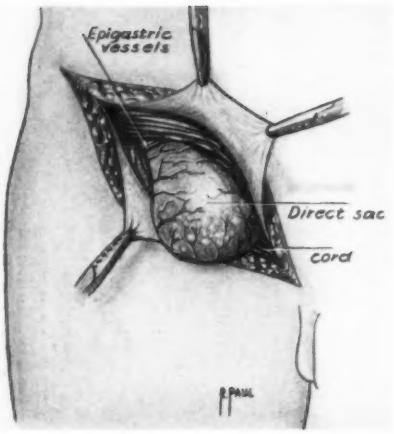

Fig. 3. Appearance of the inguinal floor in a direct hernia in which the fascia transversalis is entirely missing.

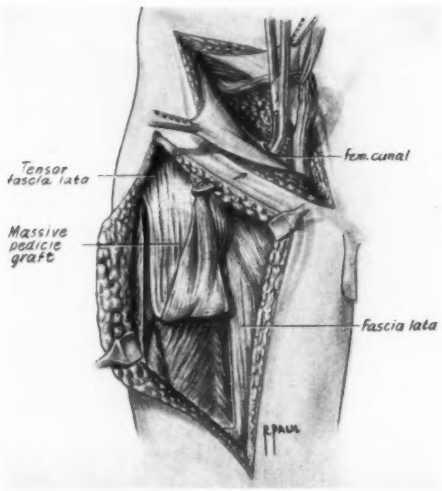

Fig. 4. First stage in the mobilization of the massive pedicle fascial graft obtained from the fascia lata. Notice that in this graft is also included part of the tensor fascia lata. The curved hemostat has been passed through the femoral canal into the subcutaneous adipose layer ready to transpose the graft into the inguinal region.

2. Fascial sutures (*Gallie and McArthur*). When marked relaxation of the floor is present with loss of support in the inguinal triangle because of attenuation of the transversalis fascia, associated with dilated, incompetent internal ring and diminished muscular tone, strips of fascia lata have been used to reinforce the transversalis fascia and to insure more certain union. If only one suture is considered adequate, a strip is taken from the aponeurotic edge as has been suggested by McArthur. In 25 recurrent hernias followed 3, or 12 per cent, recurred.

3. Attachment of transversalis fascia to Cooper's ligament (*6*). In cases in which a locule of the sac protrudes into the femoral canal, a femoral hernia, or in which Poupart's ligament is shallow with narrow shelving portion or tends to tear easily, the transversalis fascia should be attached to Cooper's ligament by silk or fascial sutures. Of course, when this technique is used the fascial components must be competent. In 7 hernias repaired by us by means of this technique there has been 1 recurrence, or 14.3 per cent.

4. Pedicle fascial graft (*modified Wangensteen*). This operation is a much more formidable procedure than the ones heretofore described and has been employed only in the large intractable hernias in which other more conservative procedures have been unsuccessful. The majority of the patients in this group had been previously operated upon two or three times and had become pretty well reconciled that further operative attempts would be useless. Furthermore, some of the patients had been advised by surgeons of wide experience that their hernias were irreparable. Fortunately these difficult hernias occurred only 8 times in our series of 130 recurrent cases, an incidence of 6.0 per cent.

On examination of the inguinal region one finds a huge hernia, usually containing coils of intestines, which is easily reduced. On palpation there is noted an elongated, oval defect in the wall just medial and parallel to Poupart's ligament, admitting perhaps three or four fingers without meeting any supportive structure in the floor. The all important fascia is not demonstrable. At operation after the muscular and fascial planes are mobilized, it is quite obvious that the fascia transversalis is either missing or perhaps only a medial remnant of it is left so that it can no longer be made to span the defect by suturing it to the inguinal ligament. Then too, in these large and usually long standing hernias there is concomitant general relaxation and weakness of all the tissues in this region. The latter are secondary contributing factors that must be included in appraising the indications for a radical hernioplasty.

TECHNIQUE OF OPERATION

After the scar in the inguinal region is excised, the sac is evacuated of its contents (Fig. 3). All

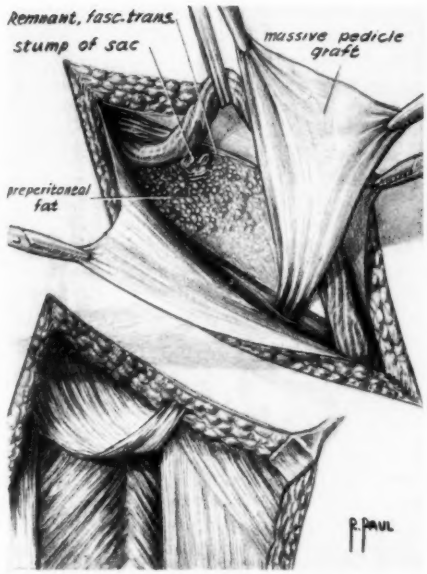

Fig. 5. Second stage in the mobilization and transposition of the graft. The graft has been already transposed into the inguinal region via the femoral canal.

loculles are freed, converted into one, and the wall of the bladder, which is usually adherent, is dissected away, thus permitting high ligation of the sac. The muscular and fascial planes are then identified, and the cord is mobilized so that a clear picture of the extent and cause of the defect is evident. The next step is to raise a flap of fascia lata 5 to 6 centimeters in width. Up to this point the details have been identical with those described by Wangensteen (Fig. 4), but at this juncture they differ from the latter in that a curved forceps is carefully insinuated through the femoral canal (Figs. 1 and 4), grasping the fascia and drawing it through the canal (Figs. 1 and 5), without rotating it, to avoid any circulatory impairment which might interfere with its viability. The fascia is then fanned out and sutured to the margin of the defect (Fig. 6), i.e., remnant of transversalis fascia, the transversalis and internal oblique muscles opposite the internal ring at the exit of the cord and laterally to the deep shelving portion of the inguinal ligament and to Cooper's ligament (Figs. 2 and 6) at the posterior arc of the femoral ring. Thus the surfaces of the transposed graft assume a converse relation. The superficial side which may have a few attached fat globules comes to lie in contact with the preperitoneal fat, and the smooth, glistening, deep surface becomes apposed to the fascial and muscular lamina. The aponeurotic flaps are then overlapped beneath the cord (Fig. 7). No attempt is made to close the fascial defect in the thigh.

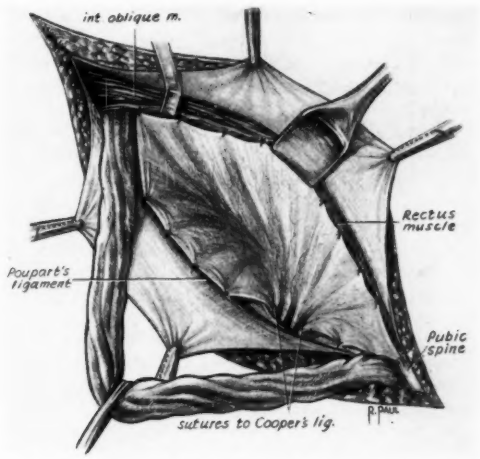

Fig. 6. Last stage in the fixation of the graft to supplant the missing fascia transversalis. Notice the deep stitches fixing the graft to Cooper's ligament.

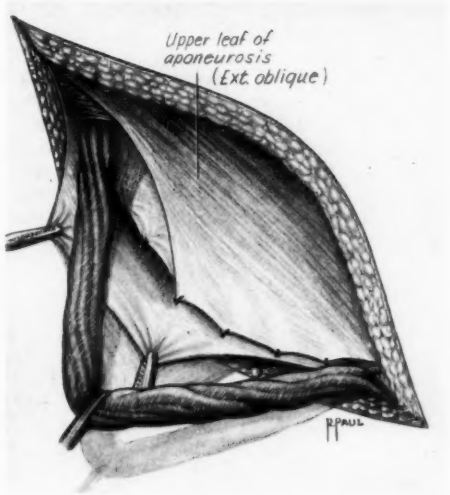

Fig. 7. Further stage in the repair of the hernial defect. The upper flap of the aponeurosis of the external oblique has been attached in part to the shelf and overshelf of Poupart's ligament. In large defects this might not be possible.

Because of the large area of denuded and exposed tissue, rigid asepsis and meticulous hemostasis must obtain as infection or the formation of a hematoma would seriously jeopardize a favorable outcome.

The advantages of the modified Wangensteen technique are: (a) the pedicle fascial graft is more deeply placed, occupying the same anatomical level as the former fascia transversalis; (b) it reaches this position through a normal patent canal without compromising the function of the inguinal ligament; and (c) it obliterates the femoral canal which prevents any chances of the formation of a femoral hernia.

In our series this type of repair has been performed fifteen times. Seven of these have been done during the past year so that only 8 are included in the follow-up. The original Wangensteen technique was followed in the first 2 cases, bringing the pedicle flap over the inguinal ligament. However, it seemed to us that a more secure wall would be obtained if the flap were more deeply placed. Accordingly, 6 operations were done in which the femoral canal was used as the passageway for the fascial flap. In this group there has been one recurrence which occurred in the original or anterior flap transposition but thus far there have been no recurrences in those cases in which the transfemoral canal method was used. Infection occurred in 1 case which drained for several weeks until the silk sutures were extruded, but this did not preclude the obtaining of a strong wall. In none of the cases in this group has there been hemorrhage, thrombophlebitis or any demonstrable circulatory impairment of the extremity from trauma or proximity of the graft to the femoral vessels. The defect in the fascia lata has caused no complaints of weakness but there has been slight bulging of the lateral thigh muscles and an occasional small area of paresthesia has been detected.

TABLE III.—FOLLOW-UP STUDIES

Time in months	Hernias followed up	Recurrences	Percentage recurrence
6 to 12	10	3	2.9
13 to 24	39	5	4.9
25 to 36	20	3	2.9
37 to 48	26	3	2.9
49 to 54	6	0	0.0
Total	101	14	13.6
Seen by physicians	65	8	....
Letter contact	36	6	....

Table III gives an analysis of the cases which have been followed up. The greater percentage of recurrences occurred in the first 3 years (78 per cent of the total).

SUMMARY AND CONCLUSION

A statistical survey is presented of 130 reoperated upon recurrent hernias. One hundred and one cases were followed up for a maximal period of 54 months of which 76 per cent were re-examined by physicians. So far there have been 14 recurrences, or 13.6 per cent of the total followed up.

The causes for failure in the original repairs have been discussed from the standpoint of the sacs and the defects in the walls, respectively. The most striking finding in regard to the sacs was the marked preponderance of the direct type in 54.7 per cent, bilocular in 20.7 per cent, and diverticular in 13.8 per cent of the recurrent hernias. The importance of the integrity of the fascia transversalis has been emphasized.

A modification of the original Wangensteen technique has been described and illustrated—a pedicle graft of iliotibial tract fascia is transposed through the femoral canal. We feel that this type of radical hernioplasty replaces with a strong viable, sheet-like structure the all important fascial floor more deeply than does any other type of operation. Six patients operated upon with exclusive adherence to this modification were followed for over 30 months and there have been no recurrences. Seven patients operated upon in the past year have so far shown every evidence of successful results.

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EDITORIALS

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PROTRUDED INTERVERTEBRAL DISCS

THE results of the surgical removal of protruded intervertebral disc during the past few years have established the syndrome on a sound anatomical, clinical, and pathological basis. Repeated demonstrations in hundreds of instances have shown that protrusion of one or more intervertebral discs into the spinal canal is a common cause of intractable backache and sciatic pain. The demonstration of this syndrome is a memorable advance in the elucidation of the problem of chronic backache.

Backache annually results in untold pain and suffering and in a tremendous loss of man power. It is a complaint that is at times extremely difficult to explain and often results in knotty medicolegal battles.

Until the condition of protruded intervertebral disc was sufficiently understood to permit satisfactory clinical recognition and radiological demonstration, many patients erroneously were considered as neurotics or pure malingerers. The recognition and correction of the pathological lesion have resulted, in a large percentage of cases, in restoration of health to the individual and in a return to his former gainful occupation.

Our present knowledge concerning protruded intervertebral discs has been built up from the contributions of many contemporary writers. The earlier writers, although unacquainted with the lesion as it is known today, laid the foundation for many of the principles which are now employed in the diagnosis and treatment of this relatively common intraspinal lesion. The writings of Elsberg, Goldthwaite, Cushing, Sicard and Forestier, Adson, and Dandy contributed in large measure to the present understanding of obscure, intraspinal, space taking lesions. These authors, along with others, devised the means of diagnosing lesions which do not give objective neurological evidence of their presence or their exact location. These men laid the foundation for the successful surgical attack on such lesions.

The demonstration by Goldthwaite emphasized the great importance of intervertebral disc in the production of backache, sciatica, and paraplegia. His valuable paper was an outstanding contribution, but unfortunately had to be rediscovered after a great deal of the more recent work had been done. The introduction of pneumomyelography and later of fluoroscopic examination of the subarachnoid space by means of radiopaque oil (lipiodol) was really the tool which led to the recognition of the fact that protruded intervertebral disc is not a rarity but a relatively common occurrence. When the lesion had been seen radiologically and had

been demonstrated repeatedly at the operating table it was only a step to discover that it was possible, by careful analysis of large series of cases, to correlate the symptoms and findings and to establish certain criteria which constitute the "disc syndrome."

The syndrome has become so well established that in the vast majority of cases the true nature of the underlying lesion accounting for intractable backache or sciatica or both can be recognized after a carefully taken and considered history and examination. Roentgenological examination by means of contrast medium is for the most part only confirmatory evidence. The diagnosis of this lesion, which was formerly considered rare, has been made relatively simple in the majority of cases since the frequency with which it actually occurs has been recognized. The method of treatment generally accepted by those who have had most experience with it suggests that the operative removal of protruded discs is a highly technical procedure and should be undertaken only by those trained to perform operations on the spinal cord.

The surgery of protruded discs has advanced rapidly and in keeping with the great progress made in the diagnostic field. The earlier operations were extensive and time consuming and necessitated a long period of hospitalization and convalescence. As knowledge of the clinical behavior, site of predilection, and pathology of the protruded disc and of the associated lesions (such as thickening and fibrosis of the ligamentum flavum) has developed, the operative removal of the protrusion has been simplified considerably as far as the patient is concerned. The simplified procedure demands considerable experience on the part of the surgeon. Most intraspinal intervertebral protrusions of discs today can be removed without the interruption of a single neural arch. Many can be removed without the sacrifice of any bone of the spinal column. This, of course, leaves the spinal column as nearly normal as possible, in fact, postoperative roentgenograms of a spinal column will fail to disclose any change not seen in the pre-operative roentgenograms. This is an important consideration, particularly in medicolegal and compensation cases.

The results of operative removal of protruded intervertebral discs as has already been stated are, in the majority of cases, highly successful. The removal of the protruded part of the disc results in relief of disabling pain, and restoration of the individual to his former occupation.

The selection of cases, the type of treatment the patient has had previously, the accuracy with which the lesion is removed at operation, the postoperative care, and the question of insurance and compensation are important factors in determining the final result.

J. GRAFTON LOVE.

DISTENTION ASSOCIATED WITH SPREADING PERITONITIS FOLLOWING PERFORATIONS OF ABDOMINAL VISCERA

THE majority of physicians believe that abdominal distention is a sign which invariably accompanies spreading peritonitis. Authors of textbooks on surgery have maintained and medical students have been taught that it must be present before this diagnosis can be made.

Approximately 85 per cent of the deaths from spreading peritonitis follow perforation of the appendix, duodenum, gall bladder, or diverticula of the colon. Pelvic inflammatory disease, contamination of the peritoneum during or following operation, perforated malignant and non-malignant lesions of the stom-

ach, perforation of gangrenous intestine, a Meckel's diverticulum, perforated typhoid and tuberculous ulcer of the small intestine, traumatic rupture of viscera, perforation of the esophagus and uterus, are responsible for the remaining 15 per cent.

The mortality of acute and subacute perforations is dependent upon early hospitalization. Acute perforations are usually admitted early but they comprise only a small percentage of the total number. With the exception of perforation of the duodenum, frank perforations in the alimentary tract are rare. The sudden escape of a portion of the contents of the duodenum and stomach into the greater peritoneal cavity immediately produces irritation of the visceral and parietal peritoneum and the large nerve plexuses, resulting in agonizing morphine resistant pain, shock, and persistent rigidity. The diagnosis is invariably made and the patient consents to immediate hospitalization—he knows something catastrophic has occurred.

The majority of perforations are subacute and consequently develop slowly. Usually more than 24 hours elapse between the initial involvement of the mucosa with subsequent gangrene and perforation of the serosa. During this period, changes which are protective in character occur in the tissues in close proximity to the potential perforative site. These changes so limit the absorption of bacterial toxins that when perforation actually occurs, the plexuses of nerves responsible for the institution of defensive measures are only moderately stimulated. Pain may be only slightly or moderately increased with no evident change in the physical signs. The one exception is a subacute perforation converted into an acute one by peristalsis induced by a laxative or some other drug.

Distention of the abdomen associated with spreading peritonitis may be extraluminal or intraluminal, local or general, and may be due to gas or fluid, or both. Extraluminal gaseous distention of the abdomen is uncommon but occurs locally in acute perforations of duodenal ulcers. Early evidence of this may be shown by a roentgenogram. Later, the presence of gas can be demonstrated by percussion and measured as it increases. If the patient is seen within 2 hours after the onset of pain, and the lower border of liver dullness is marked on the epidermis, reexamination each succeeding 2 hours will show a step-ladder diminution. During this time peristaltic sounds will be absent and the lower half of the abdomen will show no evidence of distention.

Neither general nor local gaseous distention is an early sign of acute perforation of an acutely inflamed appendix. A gradual distention of the cecum and terminal ileum is evidence of the localization of a spreading process. General distention is usually indicative of a spread of the process.

Acute perforation of the gall bladder resulting in early spreading peritonitis is rare; subacute perforations are comparatively frequent, but the process is so completely quarantined that symptoms and signs are few. Distention of stomach and transverse colon may occur; general distention is unusual.

Perforation of a diverticulum of the sigmoid is seldom sufficiently acute to cause a fatal spreading peritonitis. If a laxative has not been administered, subacute perforations are preceded by the development of immunity in contiguous tissue; small abscesses are frequently absorbed; adhesions or fistula to the pelvic viscera often occur in the female, fistula to the bladder in the male. During the localization of these processes, physical examination or a roentgenogram frequently reveals a gradual increase in the diameter of the lower colon, at times of the adjacent ileum.

Of the remaining perforations which produce spreading peritonitis, rupture of the gangrenous intestine following acute obstruction is the most frequent. Perforation of the devitalized intestine usually occurs several days after the development of the obstruction. If the patient has been under observation and the distention from the obstruction has been controlled by the suction catheter, secondary distention may follow perforation but invariably the patient's reactive capacity is so low that this sign may be very slight or entirely absent. The exception to this is the sudden rupture of gangrenous intestine due to a subcutaneous or intramuscular injection of eserine or pituitrin.

Acute perforations of typhoid or tuberculous ulcers may be followed by early general distention; such perforations, however, are more often subacute and as they frequently occur in hospitalized patients a surgical opinion regarding management is sought comparatively early. A small percentage of these patients show air in the peritoneal cavity, as do acute blow outs in the intestine.

Patients die of spreading peritonitis because absorbed toxins have not been adequately neutralized. In the acute perforations, and to a lesser degree in the subacute, Nature's initial protective measure is the induction of intra-abdominal quietude; peristalsis ceases, the abdominal muscles contract, and the excursions of the diaphragm are limited. These measures, however, are only adjuvants to the processes which actually protect—processes which result in the development of local and general immunity.

Immediately following acute perforation absorption of the peritoneal contaminant begins. This is accompanied by the transudation of serum and the migration of neutrophils. Later the larger macrophages, mononuclears, and clasmatocytes appear and the serous coat may be covered with fibrinous exudate. In the subacute perforation, hyperemia, edema, and peritoneal transudate may precede actual perforation. Recent investigations have shown that in a definite percentage of cases this fluid contains antibodies.[1] With the exception of frank duodenal perforation the preceding describes what occurs in the average case. The process is usually limited to a quadrant of the abdomen. Distention is not a significant sign. Coils of moderately distended intestine may be observed but juxtaposed will be loops of normal or contracted intestine, the tone and color of which indicate that a stage of compensation exists.

Early gross evidences of decompensation are the replacement of the normal pink color of the intestine by a dusky hue, loss of tone, and increase in distention—all are the result of toxins acting on the myenteric plexus.

General gaseous distention is equally as much a late sign of spreading peritonitis as a running pulse and cold, clammy skin—all indicate the failure of neutralization of toxins.

JOHN O. BOWER.

[1]Titration of the exudate from the peritoneal cavity of patients operated upon for acute non-perforative appendicitis shows antitoxin to the Clostridium welchii in 33.33 per cent of cases.

EARLY AMERICAN MEDICAL SCHOOLS

THE SPECIAL SCHOOL OF MEDICINE OF THE LOUISIANA STATE SEMINARY OF LEARNING

WILLIAM DOSITÉ POSTELL, M.S., B.S. in L.S., New Orleans, Louisiana

LOUISIANA State University owes its origin to certain land grants made to Louisiana by the United States Government in 1806, 1811, and 1827 "for use of a seminary of learning." The State Constitution of 1845 provided for the sale of these lands and for the establishment of the institution with the proceeds. In 1848 a Board of Visitors was appointed, whose duty it was to draw up a course of study, arrange for the accommodation of students, and engage a superintendent and faculty. In 1853, after considerable discussion, a site in Rapides Parish, some 3 miles from the present town of Alexandria, was selected, and a suitable building was erected. William Tecumseh Sherman was appointed superintendent, 5 professors were engaged, and the instruction of 19 students was begun January 2, 1860.

A more unpropitious time could scarcely have been chosen. The first session lasted until July 1, 1860, and the second opened November 1. Before it had ended the War between the States broke out, Superintendent Sherman resigned to enter the Union Army, and practically the entire faculty and student body departed immediately afterward to enter the Confederate Army. The Louisiana Seminary of Learning struggled along until 1863, when the Red River Valley was invaded by the Union Army, but finally closed its doors April 23 of that year.

David F. Boyd in the uniform of a Confederate soldier. Reproduced from a daguerreotype made during the War between the States.

Academic work was resumed October 2, 1865, with Major David French Boyd as acting superintendent. He had been appointed to that position earlier in the same year by James Madison Wells, Governor of Louisiana, just after the surrender of the Confederate forces in the Northern part of the state. Major Boyd (like most of the first superintendents of Louisiana State University he soon became "Colonel") had been a member of the original faculty and had resigned to enter the Confederate Army. He served as superintendent until 1880 and was reappointed for a two-year term in 1886.

In 1869 the Seminary building was destroyed by fire, but activities were immediately transferred to Baton Rouge, where the main plant has been located ever since. In 1870 the name of the Louisiana State Seminary of Learning was changed by Legislative Act to Louisiana State University. Later the University was merged with the Agricultural and Mechanical College which had been opened in 1874 in New Orleans, and the two state institutions, "united and constituted into one and the same institution of learning," began their joint operation October 5, 1877.

Colonel David French Boyd, who reopened the University after the War between the States and who guided it through the difficult days of reconstruction, early envisioned the Seminary, as it was then called, as a great state university "where literature, science and all the arts may be taught; where the principles of truth and honor may be established,

Librarian of the School of Medicine of Louisiana State University.

and a noble sense of personal and patriotic and religious duty inculcated, in fine, to fit the citizen to perform justly, skillfully, and magnanimously all the offices, both private and public, of peace and war."[1]

The right and duty of Louisiana State University to operate a school of medicine was specifically set forth in the Legislative Act of 1877 which is the charter of the present institution. This charter provides for[1]

"general instruction and education in all the departments of literature, science, art and industrial and professional pursuits . . . special instruction for the pursuits of agriculture, the mechanical arts, mining, military science and art, civil engineering, law, medicine, commerce and navigation."

But even before provision for a school of medicine had been made by legislative act, steps had been taken to provide medical training in the Louisiana State Seminary of Learning. June 11, 1866, according to the minutes of the Board of Supervisors, a Mr. Boyce had introduced the following resolution:[2]

"Whereas a general knowledge of Anatomy, Physiology and Hygiene is desirable in the students of this institution there be created a professorship of these branches, with a salary of one thousand dollars per annum with quarters and that the Surgeon of the Seminary shall be the professor of the said chair."

Previously, in February of that year, the vice-president of the Board had been authorized to advertise in the newspaper for a surgeon[2]

"who would also serve as professor of Chemistry, Geology and Mineralogy . . . who shall be a graduate of medicine and an experienced physician; his testimonial embracing qualifications in both branches."

The necessary qualifications had been found in Dr. J. W. Wilson, a relative of the then governor of Louisiana, Henry W. Allen, and he had accordingly been elected acting surgeon of the seminary. Dr. Wilson, who was a native of Virginia, had practiced medicine for 15 years in St. Louis before entering the Confederate Army, but could not return to Missouri immediately after the War because of the test oath requirement of that state. The day after the resolution authorizing the establishment of the special school of medicine had been passed, he was notified of his appointment as surgeon and professor of anatomy, physiology and hygiene as follows:[2]

"Louisiana State Seminary of Learning and Military Academy

June 12, 1866

"Dr. J. W. Wilson

"Surgeon, Louisiana State Seminary of Learning and Military Academy

"Sir: I am instructed to inform you that the Board of Supervisors of the institution at its stated annual session yesterday chose you as the permanent Surgeon of the institution your compensation as such to be the medical fee of one dollar per month from each cadet in the institution, until the same shall be $2000. You were also chosen professor of Anatomy, Physiology and Hygiene in the institution with a salary of one thousand ($1000) dollars per annum—and quarters in the capacity of Surgeon and Professor with the understanding that until there shall be full employment for your time in your own especial department you will discharge such duties (the next word is illegible in the handwritten document) to the welfare and interest of the institution as you may be qualified for and may be assigned to by the Superintendent.

"Hoping to hear from you soon that it will be agreeable to you to accept the positions to which you have been chosen with the salaries and conditions attached thereto, I am

"Very respectfully

"G. Mason Graham

"Vice-President of Board of Supervisors Louisiana State Seminary of Learning and Military Academy."

The Board also appointed Dr. John R. Page to the faculty of the special school of medicine to give the lectures in materia medica. Dr. Page, who, like Dr. Wilson, had served in the Army of the Confederacy, was a graduate of the University of Virginia.

The course of study, according to the catalogue issued in 1866,[3]

"embraces a preparatory and an academic department and two special schools, one of Civil Engineering and the other of Medicine . . . and of the School of Medicine its purpose is to give a good preliminary preparation for the School of Medicine in New Orleans. *But more accomplished.* In both the special schools, the French and Spanish languages, Belles Lettres, Mental and Moral Philosophy and Political Economy are taught; in the School of Medicine the study of Latin is pursued, and Anatomy and Physiology are studies in the School of Civil Engineering."

The School of Medicine in New Orleans was then the medical department of the University of Louisiana and is now the School of Medicine of Tulane University. The proposed instruction was to cover 4 years, the first 2 to consist of liberal arts courses and the last 2 to consist of specialized training in medicine. An insight into the educational ideas of the times is gained by certain provisions in the curriculum. Thus materia medica, mineralogy and geology were taught as a single course, and it will be observed

[1] Acts Passed by the General Assembly of the State of Louisiana at the First Session of the Fifth Legislature Begun and Held in the City of New Orleans January 1, 1877 and at the Extra Session Convened at the City of New Orleans March 2, 1877. New Orleans, George W. Dupre and Company, 1877. Act No. 145.

[2] Minutes of the Board of Supervisors of the Louisiana State Seminary of Learning and Military Academy, 1865–1876.

[3] Official Register of the Officers and Cadets of the Louisiana State Seminary of Learning and Military Academy Session Ending June 30, 1866.

SPECIAL SCHOOL OF MEDICINE

Junior Class—First Year

Studies	Professor	Texts
Anatomy, Physiology	Prof. J. W. Wilson, M.D.	Wilson's Anatomy and Carpenter's Physiology
Chemistry	Prof. J. R. Page, M.D.	Fowne's Chemistry, Leebig's Agriculture Chemistry
French	Prof. J. P. Bellier Ass't Prof. R. E. Rives	Telemachus, Histoire de France
Latin	Prof. D. F. Boyd	Horace, Livy

Curriculum for the Junior class in the Special School of Medicine, Louisiana State Seminary of Learning and Military Academy, Alexandria, La., 1866.[1]

SPECIAL SCHOOL OF MEDICINE

Senior Class—Second Year

Studies	Professor	Texts
Anatomy, Physiology	Prof. J. W Wilson, M.D.	Wilson's Anatomy and Carpenter's Physiology
Materia Medica, Mineralogy and Geology	Prof. J. R. Page, M.D.	U. S. Dispensatory, Dana's Mineralogy, Hitchcock Geology, Practical instruction in assaying ores and alloys, smelting metals and analyzing soils.
Spanish	Prof. J. P. Bellier Ass't Prof. R. E. Rives	Butler's Spanish Teacher
Moral Philosophy	Prof. D. F. Boyd	Whatley's Logic, Paley's Moral Philosophy
Belles Lettres	Ass't Prof. J. M. Boyd	Butler's Analogy

Curriculum for the Senior class in the Special School of Medicine, Louisiana State Seminary of Learning and Military Academy, Alexandria, La., 1866.[1]

that Latin was a required course in the school of medicine, while anatomy and physiology were required in the school of civil engineering.

The catalogue further states:

"The graduates in the special schools will receive diplomas with the title 'Graduate in the School of Civil Engineering' and 'Proficient in the School of Medicine.'"

One of the interesting activities of the medical school was the early attempt to build up a medical library. According to Bulletin No. 20 of the United States Bureau of Education the embryo library contained 443 books of medicine and received the following scientific periodicals: *Eclectic Magazine*, *Journal of Science and Art*, *Journal of Pharmacy*, *Popular Science Monthly*, *Annales de Chimie*, and the *Scientific American*.[2] Apparently all that remains of the original collection is a three-volume set of *Leçons orales de clinique chirurgicale*, which is now in the Agramonte Memorial Library of the present school of medicine of Louisiana State University. The stamp on its title page, "Louisiana State Seminary of Learning and Military Academy near Alexandria, La.," is evidence of its inclusion in the earlier library.

Little seems to have been accomplished during the session 1866–1867 toward developing the special school of medicine and the authorities apparently realized almost at once that their efforts had been ill timed and premature. A resolution was introduced in October, 1867, recommending that it be discontinued, but it was decided that action be deferred until the plan had been given a longer trial. The 1867 catalogue, however, makes no mention of the medical school, and the minutes of the board of supervisors show that Drs. Wilson and Page were assigned to teach other subjects.

So ended the first abortive courses in medicine taught at Louisiana State University. In spite of the specific provision in the Charter of 1877[3] for the establishment of a medical school, nothing was done in that direction until December, 1930, when plans to open the present school in New Orleans were made. The first students were admitted in the Fall of 1931, the newly erected building on the grounds of the Charity Hospital of Louisiana at New Orleans was occupied in the spring of 1932, and the first 4-year class was graduated in June, 1935. There is no doubt that Colonel David French Boyd, who is chiefly responsible for the University as it exists today, would be more than pleased with the establishment at long last of the school which he had tried to establish almost 75 years earlier.

[1] See footnote 3 on preceding page.

[2] Edwin Whitfield. History of Education in Louisiana. U. S. Bureau of Education. No. 20. Government Printing Office, 1898. Pp. 93–94.

[3] See first footnote on preceding page.

THE SURGEON'S LIBRARY

REVIEWS OF NEW BOOKS

THE treatise, *The Rectum and Colon*[1] by E. Parker Hayden, on the common complaints of the lower portions of the intestinal tract is designed primarily for the general practitioner. Based chiefly upon the writer's personal experience, it omits material of a controversial nature, which would be of academic interest mainly to the specialist. Because it describes treatment and surgical technique which have given excellent results in the author's hands, presented in a simple, understandable, and effective manner, the book is recommended as a dependable guide for those who are called upon to relieve these disorders. Especially practical is the chapter devoted to a consideration of symptoms, since a thorough knowledge of the pathological physiology allows a more successful application of the principles used in diagnosis and treatment.

The book is well written, nicely proportioned as to subjects and their respective importance and is clearly printed and illustrated with good plates and photographs, which are sufficiently helpful but not so profuse as to make the cost excessive. It will be of value to anyone wishing more complete information on proctological and colonic troubles, and physicians undertaking surgery of these parts should find it a definite aid. DURAND SMITH.

THE book, *Nitrous Oxide-Oxygen Anesthesia*,[2] by F. W. Clement, represents a thorough compilation of material on this subject. It presents the teachings and findings of Dr. E. I. McKesson, who developed the technical administration of nitrous oxide-oxygen to the point where it could be used on all types and conditions of patients, for every requirement and emergency of surgery and anesthesia, and every operative procedure in both the general surgical and dental fields. It also presents the author's own impressions and experiences resulting from the use of nitrous oxide-oxygen over a period of many years.

The book is well written and easily read. It is printed on a fine grade of paper, is well bound and illustrated, and comprises 268 pages.

The chapters on the physiology of anesthesia are especially well presented. It charts a classification of signs and symptoms of nitrous oxide-oxygen anesthesia and discusses the fallacy of cyanosis perse as a true sign of nitrous oxide-oxygen anesthesia. The administration of nitrous oxide-oxygen is discussed in detail with emphasis on the primary and secondary saturation techniques, depression tests, positive method of anesthesia, endotracheal anesthesia, the advantages and disadvantages, and comparison with other gaseous anesthetics used.

Some of the principles brought out are contrary to those practiced by many other anesthetists, especially the technique of secondary saturation which is considered by many to be fraught with the dangers of asphyxia.

As a guide and reference book for the physician anesthetist, this excellent work can be most highly recommended. MARY KARP.

AS the title implies, Professor Jamieson's *Illustrations of Regional Anatomy*,[3] is a pedagogical offering in a series of pictures of gross human structure; there is no text accompanying and no directions designed to guide the dissector; even the legends are of the briefest order.

The illustrations are grouped in 7 short volumes. In each of the sections the pages are fitted on to loose-leaf pillars from which they are readily removable for rearrangement in any sequence which meets the current need of the student. The pages are printed on one side only, so that they may be pasted into notebooks.

The 305 illustrations cover the subdivisions of the body: central nervous system, head and neck, abdomen, pelvis, thorax, upper limb, and lower limb. The head and neck are allotted the largest number of figures, the thorax the smallest number.

The brain is extensively illustrated in sections; basal nuclei, motor, and sensory areas are indicated diagrammatically, as are also the principal tracts, commissures, etc. Blood vessels, cerebral ventricles, and choroid plexuses are all presented with profuse coloring.

The head and neck are illustrated by drawings which include entire dissection fields, layered dissections of individual sense organs, diagrammatic representations of nerve plexuses, and transverse and sagittal sections. Some of the figures would be baffling to a beginner, since they do not indicate what has been excluded, or which layers have been reflected in order to bring into view the stratum which the figure portrays. Others lose value by being too courageously diagrammatic, suggesting but not closely resembling the structure as it appears in the human body.

The section on the abdomen contains ingeniously diagrammatic illustrations of the abdominal muscles

[1] THE RECTUM AND COLON. By E. Parker Hayden, A.B., M.D., F.A.C.S. Philadelphia: Lea & Febiger, 1939.

[2] NITROUS OXIDE-OXYGEN ANESTHESIA; MCKESSON-CLEMENT VIEWPOINT AND TECHNIQUE. By F. W. Clement, M.D. Philadelphia: Lea & Febiger, 1939.

[3] ILLUSTRATIONS OF REGIONAL ANATOMY. By E. B. Jamieson, M.D. Sections I–VII. 2d ed. Baltimore: Williams & Wilkins Co., 1939. (American distributor.)

and the inguinal canal, but some of them sacrifice anatomical clarity for schematic vividness. The treatment of the inguinal region, mesenteries, blood vessels, kidneys, and suprarenals is highly conventional, resembling models rather than dissections. The schematic drawings of abdominal layers are architectural achievements but take considerable liberty with the real fabric of man's body.

In the volume on the pelvis and perineum color is again lavishly and helpfully used, but the author's order of plates is seemingly quite haphazard for a section of anatomy in which a knowledge of progressive stratification is of the greatest importance. While the layers of the pelvic and urogenital diaphragms are shown in schematic clearness, they are not portrayed accurately; fascial layers of the perineum are shown in disjunction, not as strata continuous with layers in adjacent regions; the relations of the pelvic viscera to their ligamentous, fascial, and diaphragmatic supports would be of little use in training critical students for the practice of gynecology, urology, or proctology. The figures illustrating the anatomy of the female pelvis are the most unfortunate of the entire lot.

In the set of plates portraying the structure of the thorax, the schematic method is utilized to greater advantage, since much of the important anatomy of the thoracic cage and its viscera may be taught from transverse and sagittal sections and from surface projections of organs.

In the sections on the upper and lower limbs, the figures which show the areas of muscular attachment on the bones are fundamentally similar to those found in the standard textbooks, atlases, and manuals of gross anatomy. Here the order is the logical one of successive layers, from cutaneous nerves, superficial veins, and fascial sleeves, through progressively deeper layers of muscle. In the more simple figures, for example, those dealing with the synovial sheaths of the hand and foot, the schematic plan is utilized to great advantage; but when the same device is employed to show the tendons, nerves, vessels, and muscles, the collection of transected structures forms a bewildering assemblage.

The colors employed in the illustrations seem, in some instances, unnecessarily garrish; but the reader cannot blame either the author or illustrator for indulging in such innocent gaiety, when chromatic feasts of this kind are so rare in the history of bookmaking. Continued examination of the plates lowers one's opposition to the resplendent use of color, since it is realized that the varied hues function as ocular signals, for what student would ever fail to remember the tripartite character of the innominate bone, after he has seen its units done in gray, cerise, and cerulean blue?

Taken as a whole, *Illustrations of Regional Anatomy* is a useful adjunct to the student's regular anatomical library; it treats of the kind of anatomy which is customarily presented in charts. Its graphic character is analytical of the standard type illustration or take-apart models found universally in present day laboratories. The volumes of figures will serve as a link between the standard atlas which the student uses at the dissection table and the blackboard drawings which are customarily a feature of the lecture in gross anatomy. BARRY ANSON.

IN a treatise of 400 pages entitled *Sterility and Impaired Fertility*[1] Lane-Roberts presents the subject in a comprehensive manner. The work covers this important problem of the obstetrical and gynecological field in a detailed fashion. Each phase of it is given thorough consideration. The male factor is given a place equal to that of the female, and it is encouraging to find that this phase of sterility is being stressed as it should be. Anomalies in the male genital tract, difficulties resulting from physiological disturbances, methods of collecting and examining semen, and evaluation of the results obtained are clearly set forth.

In considering the female's rôle in sterility, the human cycle is reviewed. Illustrations of the endometrium at different stages of the cycle are included. The endocrines and disturbances of the endocrine mechanism are discussed in relation to the human cycle.

Under the investigation of "specific functions," ovulation and methods for the determination of its occurrence, the patency of the tubes, endometrial changes, and tests for determining the state of the endocrines are discussed. The rôle of hormones in the production of sterility and their uses in its treatment are well presented.

This volume is recommended to anyone who is interested in the subject of sterility.

CHESTER C. DOHERTY.

THE title of the excellent 165 page monograph, *Untoward Effects of Nitrous Oxide Anesthesia*,[2] is somewhat misleading. It deals, in fact, with the untoward effects of anoxia which may accompany nitrous oxide anesthesia. It calls attention to the morbidity and mortality following nitrous oxide which have only recently been understood although they have long been observed.

The author, Cyril B. Courville, has conducted a competent and painstaking investigation into the neuropathology found in victims of nitrous oxide administration, based on 26 cases which have come under his observation. Case histories and postmortem findings are presented.

The pharmacology and toxicology of nitrous oxide are discussed. The important fact is noted that deep surgical anesthesia with nitrous oxide is obtained only with asphyxia. A complete review of the neuro-

[1] STERILITY AND IMPAIRED FERTILITY; PATHOGENESIS, DIAGNOSIS AND TREATMENT. By Cedric Lane-Roberts, M.S., F.R.C.S., F.R.C.O.G., Albert Sharman, M.D., M.R.C.O.G., Kenneth Walker, F.R.C.S., and B. P. Wiesner, D.Sc., Ph.D., F.R.S.E. With a Foreword by The Rt. Hon. Lord Horder, G.C.V.O., M.D., F.R.C.P. New York: Paul B. Hoeber, Inc., 1939.

[2] UNTOWARD EFFECTS OF NITROUS OXIDE ANESTHESIA; WITH PARTICULAR REFERENCE TO RESIDUAL NEUROLOGIC AND PSYCHIATRIC MANIFESTATIONS. By Cyril B. Courville, M.D. With Foreword by Dr. Yandell Henderson. Mountain View, Calif.: Pacific Press Publishing Ass., 1939.

pathological findings in asphyxia from various causes is presented together with certain general pathological considerations.

The work is amply illustrated. It is recommended for careful study by all those whose patients receive anesthetics, and it should be "required reading" for anesthetists.

The foreword by Dr. Yandell Henderson is a fitting introduction to the text. FRANK J. MURPHY.

THE increasing interest in industrial medicine and surgery justifies the publishing of Schwartz and Tulipan's very important book, *A Text-Book of Occupational Diseases of the Skin*,[1] for it supplies the physician's need for detailed and systematized knowledge of this subject.

Statistics show the growing importance of occupational dermatoses. In England, in 1936, there were 1,771 cases of occupational dermatitis, 84 cases of chrome ulceration, and 142 cases of occupational epitheliomas. All other occupational diseases that were compensated totaled 428 cases. It is stated by English writers that these figures do not represent the actual incidence of occupational dermatitis because only those workers who are incapacitated for 1 week or more and those who receive compensation are reported. Prosser White estimated that at least 18,000 to 19,000 cases occurred in England each year.

In 1933, 9 of our states, which kept records, reported 5,787 cases of compensated occupational dermatoses among a total of 8,875 occupational diseases, or about 65 per cent of all diseases. The latest available records from 5 of these states show that while the number of all occupational diseases has decreased in recent years, the proportion of dermatoses has increased from 65 per cent for the years 1932, 1933, and 1934 to 69 per cent in the years 1936 and 1938.

A study of the compensation records of the several states shows that the average loss of time per year for compensated cases of occupational dermatoses is about 10 weeks, and the average compensation paid is about 100 dollars. In addition there is an average cost of medical care of about 90 dollars. It is, therefore, estimated that the annual loss from occupational dermatoses in the United States is approximately 4,000,000 dollars. Most of the material in this book is based upon the authors' own observations and experiences and the remainder upon reports and observations of other workers in the field.

A very important feature of the work is a description of the various industrial processes, such as the tanning of leather, the manufacture of rubber, electroplating, the making of soap, and the manufacture of explosives. Every important industrial process is discussed briefly but so adequately that the reader is given a fuller understanding of the background on which dermatoses develop. There are interesting chapters on cosmetics, dyes, mycotic infections, occupational cancer, and diseases of the mouth. The final chapter contains a list and a detailed description of the chemicals which are known to be or which can be skin irritants.

The book contains a vast fund of information and is well written. The text is printed in large type on good paper. The illustrations are numerous and excellent, and there is an extensive bibliography at the end of each chapter. This reviewer unhesitatingly recommends it to every dermatologist, industrial physician, and general practitioner. The authors are to be congratulated on producing a book that should be a welcome addition to any medical library. EDWARD A. OLIVER.

THE 838 page English translation of Bing's fifth edition of his *Lehrbuch der Nervenkrankheiten*[2] contains 30 chapters and has 207 illustrations. The translator has rearranged and augmented the original for adaptation to American usage. Despite this the very cogent observations and conclusions of Professor Bing are retained.

The author's style of presentation is entirely in accord with that of the reviewer. If in neurology one must be methodical and accurate, it follows that the discussion of entities should be along the lines of an easy understanding, as far as possible, instead of forcing the student to memorize signs and symptoms. Professor Bing has written this book with the former in mind. All of the chapters are up to date and are written for the student; however, it is an excellent reference book for the neurologist as well. Following each section there is a very large bibliography of both American and foreign contributors. A considerable amount of discussion regarding treatment together with written prescriptions is to be found in this book. There are chapters on peripheral nerves, dyskinesias, muscular atrophies, degenerative diseases, demyelinating disorders, syphilis of the central nervous system, arteriosclerosis and hemorrhages, infectious diseases and intoxications, aphasia, apraxia and agnosia, tumors, diseases of the cerebellum, disease of the endocrine glands, diseases of the autonomic system, convulsive disorders, headaches, and psychoneuroses. The illustrations are very good. This textbook is a desirable one and is recommended to all students and neurologists. T. T. STONE.

[1] A TEXT-BOOK OF OCCUPATIONAL DISEASES OF THE SKIN. By Louis Schwartz, M.D., and Louis Tulipan, M.D. Philadelphia: Lea & Febiger, 1939.

[2] TEXTBOOK OF NERVOUS DISEASES. By Robert Bing. Translated and enlarged by Webb Haymaker. From the 5th German ed. St. Louis: The C. V. Mosby Co., 1939.

BOOKS RECEIVED

Books received are acknowledged in this department, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

FISIOPATOLOGIA EXPERIMENTAL Y CLINICA; EXPERIMENTACION ANIMAL Y EXPERIMENTACION CLINICA. By J. Pou Orfila and A. Pou De Santiago. Montevideo: Casa A. Barreiro Y. Ramos, 1939.

MANUAL DE FISIOPATOLOGIA QUIRURGICA Y CIRUGIA EXPERIMENTAL. By J. Pou Orfila and A. Pou De Santiago. Montevideo: Tipografia Atlantida, 1938.

THE FUNDAMENTALS OF INTERNAL MEDICINE. By Wallace Mason Yater, A.B., M.D., M.S. (in Med.), F.A.C.P. First edition revised. New York, London: D. Appleton-Century Co., 1940.

OBSTETRICS AND GYNECOLOGY. By The Departmental Staff of the University of Chicago and Other Contributors. Edited by Fred L. Adair, M.A., M.D., F.A.C.S., Vols. 1 and 2. Philadelphia: Lea & Febiger, 1940.

FETAL AND NEONATAL DEATH; A SURVEY OF THE INCIDENCE, ETIOLOGY, AND ANATOMIC MANIFESTATIONS OF THE CONDITIONS PRODUCING DEATH OF THE FETUS IN UTERO AND THE INFANT IN THE EARLY DAYS OF LIFE. By Edith L. Potter, M.D., Ph.D., and Fred L. Adair, M.D. Chicago: The University of Chicago Press, 1940.

TRANSACTIONS OF THE AMERICAN GYNECOLOGICAL SOCIETY. Vol. 64 for the Year 1939. Edited by Richard W. Te Linde, M.D. St. Louis: The C. V. Mosby Co., 1940.

SHOCK AND RELATED CAPILLARY PHENOMENA. By Virgil H. Moon, A.B., M.Sc., M.D. London, New York, Toronto: Oxford University Press, 1938.

CANCER IN CHILDHOOD; AND A DISCUSSION OF CERTAIN BENIGN TUMORS. Edited by Harold W. Dargeon, M.D., F.A.A.P. St. Louis: The C. V. Mosby Co., 1940.

BIOCHEMISTRY OF DISEASE. By Meyer Bodansky, Ph.D., M.D., and Oscar Bodansky, Ph.D., M.D. New York: The Macmillan Co., 1940.

CONGENITAL MALFORMATIONS; A STUDY OF PARENTAL CHARACTERISTICS WITH SPECIAL REFERENCE TO THE REPRODUCTIVE PROCESS. By Douglas P. Murphy, M.D., F.A.C.S. Philadelphia: University of Pennsylvania Press, 1940.

COMPENDIUM OF REGIONAL DIAGNOSIS IN LESIONS OF THE BRAIN AND SPINAL CORD; A CONCISE INTRODUCTION TO THE PRINCIPLES OF LOCALIZATION OF DISEASES AND INJURIES OF THE NERVOUS SYSTEM. By Robert Bing. Translated and edited by Webb Haymaker. 11th ed. St. Louis: The C. V. Mosby Co., 1940.

OXFORD MEDICAL PUBLICATIONS. TUBERCULOSIS OF BONE AND JOINT. By G. R. Girdlestone, M.A., B.M. (Oxon.), F.R.C.S.(Eng.). London: Oxford University Press, 1940.

OXFORD MEDICAL PUBLICATIONS. THE SEXUAL PERVERSIONS AND ABNORMALITIES. By Clifford Allen, M.D., M.R.C.P., D.P.M. London: Oxford University Press, 1940.

MACEWEN MEMORIAL LECTURE, 1939. THE MACEWEN OUTLOOK IN SURGERY. By G. Grey Turner, D.Ch.(Durb. Hon.), M.S., F.R.C.S., F.A.C.S.(Hon), F.R.A.C.S.(Hon.). Glasgow: Jackson, Son & Co., 1939.

THE PATHOLOGY OF INTERNAL DISEASES. By William Boyd, M.D., LL.D., M.R.C.P.(Ed.), F.R.C.P.(Lond., Dipl. Psych.), F.R.C.S. 3d rev. ed. Philadelphia: Lea & Febiger, 1940.

TREATMENT OF WAR WOUNDS AND FRACTURES; WITH SPECIAL REFERENCE TO THE CLOSED METHOD AS USED IN THE WAR IN SPAIN. By J. Trueta, M.D. With a Foreword by H. Winnett Orr, M.D., F.A.C.S. New York: Paul B. Hoeber, Inc., 1940.

ARTIFICIAL PNEUMOTHORAX; ITS PRACTICAL APPLICATION IN THE TREATMENT OF PULMONARY TUBERCULOSIS. Contributions by Saranac Lake Physicians to the Studies of the Trudeau Foundation. Edited by Edward N. Packard, M.D., John N. Hayes, M.D., and Sidney F. Blanchet, M.D. Foreword by E. R. Baldwin, M.D. Philadelphia: Lea & Febiger, 1940.

INTRODUCCION AL ESTUDIO DE LA EQUINOCOCOSIS (BIOLOGIA Y PATOLOGIA GENERAL). By Luis Mario Alonso. Buenos Aires: Librería y Editorial "El Ateneo," 1939.

EL PIE TALO POLIOMIELÍTICO Y SU TRATAMIENTO. By Dr. Vicente Sanchis Olmos. Madrid: Espasa-Calpe, S.A., 1940.

FRACTURES; AND OTHER BONE AND JOINT INJURIES. By R. Watson-Jones, B.Sc., M.Ch.Orth., F.R.C.S. A William Wood Book. Baltimore: The Williams & Wilkins Co., 1940.

ILLUSTRATIVE ELECTROCARDIOGRAPHY. By Julius Burstein, A.B., M.D. Originally written by the late Joseph H. Bainton, A.B., M.D., and Julius Burstein, A.B., M.D. 2d ed. New York and London. D. Appleton-Century Co. Inc., 1940.

RESEARCH PUBLICATIONS. ASSOCIATION FOR RESEARCH IN NERVOUS AND MENTAL DISEASE. Volume XX—THE HYPOTHALAMUS; AND CENTRAL LEVELS OF AUTONOMIC FUNCTION. Proceedings of the Association, December 20 and 21, 1939, New York. Editorial Board: John F. Fulton, M.D., S. Walter Ranson, M.D., and Angus M. Frantz, M.D. Baltimore: The Williams & Wilkins Co. 1940.

CHARLES BELL (1774–1842). Discoverer of the distinction between motor and sensory nerves; united the artist and poet with the philosopher and surgeon; an imaginative, ardent teacher with a single-minded devotion to his work; assumed direction of Hunter brothers' medical school in Windmill Street in 1812.

"*Even yet I am not rich enough to boast of my poverty.*"

Charles Bell

1774-1842